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OF

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(NUTRITION)

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EDITORIAL NOTE

The *Encyclopedia and Dictionary of Medicine and Surgery* has now reached its eighth volume, and the rubrics in this instalment extend from Physiology (Second Part) to Rhinolalia. The second half of the monograph on Physiology by Professor Noël Paton occupies nearly 100 pages, and includes a section dealing with the subject of *hormones*, which has of late assumed so great an importance. Other outstanding articles are those on Pregnancy (in eleven sections), on the Puerperium (in six sections), and on Rheumatism (in four sections). New articles have been added in which Prescribing and Methods of Administering Drugs and Radium (in its new and startling physiological effects and therapeutic uses) are fully considered. Other additions are the paragraphs dealing with the *physostigmatis semina*, *picric acid*, *potash*, and *quinine*. Some of the older articles have been revised by their authors; several new additions to the nomenclature of medicine, such as *precibiculturists*, *precipitins*, and *pro-* or *pre-maternity hospital*, have been defined; and, in a word, everything has been done to bring the work up to the level of present day theory and practice.

There are fifty-eight articles of more than 1000 words in length. These differ considerably in the space they cover, that on *Physiology* occupying not much less than one hundred pages, while for those on *Radium* and *Quinine* a space of about two pages is sufficient. The subjects dealt with in these long contributions are *Physiology* (including sections on *Blood*, *Circulation*, *Respiration*, *Digestion*, *Food*, *Hormones*, *Excretion*, and *Organic Chemistry*), *Pigments of the Body and Excreta*, the *Pituitary Body*, *Plague*, *Diseases of the Pleura* (Medical and Surgical), *Pneumonia* (in two sections, Bacteriological and Clinical), the peculiar endemic disease called *Ponos*, *Post-mortem Methods*, *Potash*, *Pregnancy* (in all its subdivisions), *Prescribing* (with a series of tables of incompatibles and of extra-pharmacopœial and other new drugs), *Prostate Gland*, *Prurigo and Pruritus*, *Psoriasis*, *Puberty*, the *Puerperium* (with its six sections dealing with the physiological and pathological developments of this epoch in a woman's life), the *Pulse*, the *Pupil*, *Purpura*, *Pyuria*, *Quarantine*, *Rabies*, *Radium*, *Raynaud's Disease and Erythromelalgia*, the *Diseases of the Rectum* (Medical and Surgical), *Refraction*, *Relapsing Fever*, *Respiration*, *Diseases of the Retina and Optic Nerve*, *Retinoscopy*, and *Rheumatism* (Acute, Chronic, in Children, and in the form of Rheumatoid Arthritis).

Of articles of less than 1000 words but of at least ten lines in length, there are sixty in this volume. These deal with such subjects as *Pica*, *Picric Acid*, *Picrotoxine*, *Pineal Gland*, *Pinta*, *Piperazine*, *Pitot's Tubes*, *Pix Burgundica*, *Placenta*, *Plasma*, *Pleurodynia*, *Pneumogastric*, *Pneumonokoniosis*, *Podophylli Resina*, *Polypus*, *Pons*, *Precibiculturists*, *Precipitins*, *Presentation*, *Prochownick Diet*, *Proteids*, *Prunes*, *Pseudocyesis*, *Ptomaines*, *Ptosis*, *Pubiotomy*, *Ulcerating Granuloma of the Pudenda*, *Pyœmia*, *Pyrexia*, *Quassia*, *Quebracho*, *Quillaiæ Cortex*, *Quinine Radical*, *Radiography*, *Râle*, *Rashes*, *Recurrence*, and *Reflexes*.

This volume also contains 919 paragraphs of less than ten lines in length. These consist of definitions of medical terms; short descriptions of processes, reactions, symptoms, and instruments; and cross references. Some of the matters dealt with in these short contributions may be enumerated: *Physiotherapy*, *Physometra*, *Phytin*, *Pia Mater*, *Piazza's Fluid*, *Pick's Bundle*, *Picrocarmine*, *Picrorhiza*, *Pigeon Breast*, *Pigeon Toe*, *Pigodidymus*, *Pillars*, *Pimenta*, *Pinard's Sign*, *Pine Oil*, *Pinguecula*, *Pink-eye*, *Pinocytosis*, *Piroplasma*, *Pithecanthropus Erectus*, *Placebo*, *Placentoma*, *Placido's Disc*, *Pladarosis*, *Plane*, *Planodia*, *Plaques*, *Plasmodium*, *Plasmon*, *Plasticine*, *Plastron*, *Platelet*, *Platycnemic*, *Plenum System*, *Pleonexia*, *Plethora*, *Plethysmograph*, *Pleurosomus*, *Plexus*, *Plica Polonica*, *Plugs*, *Plummer's Pill*, *Plutomania*, *Pneumatosis*, *Pneumomassage*, *Podagra*, *Podalic Version*, *Pododynia*, *Pogoniasis*, *Polar Bodies*, *Politzer's Bag*, *Pollantin*, *Pollex*, *Poltfoot*, *Polyadenia*, *Polycæmia*, *Polyaxon*, *Polychromatophylia*, *Polyclinic*, *Polycythæmia*, *Polygenism*, *Polymastia*, *Polymerisation*, *Polyœstrous*, *Polyphyodont*, *Polythelia*, *Pompholyx*, *Ponograph*, *Porencephalus*, *Porro's Operation*, *Porter-Clark's Process*, *Port-wine Mark*, *Position*, *Posology*, *Postclimacteric*, *Postmature*, *Potain's Syndrome*, *Potato Cream*, *Potato Culture*, *Pott's Disease*, *Poudrette*, *Pozzuoli*, *Prœcuneus*, *Prague Method*, *Prairie Itch*, *Precocity*, *Premaxilla*, *Prepotency*, *Presbyopia*, *Preston's Salt*, *Prevalidin*, *Prevost's Symptom*, *Prisms*, *Probe*, *Proctalgia*, *Pro-Maternity Hospital*, *Pronephros*, *Proptosis*, *Pro re nata*, *Prosector*, *Prosopus Varus*, *Prostatorrhœa*, *Protagon*, *Proteoses*, *Prozygosis*, *Psittacosis*, *Psoitis*, *Psorospermosis*, *Psychiater*, *Pterygium*, *Puericulture*, *Pulex*, *Pulsatilla*, *Punctum*, *Purgen*, *Purin Bodies*, *Pyrodin*, *Quercin*, *Quincke's Disease*, *Quotient*, *Rachicocainisation*, *Radioscopy*, *Railway Spine*, *Rasch's Sign*, *Rathke's Pouch*, *Rauber's Layer*, etc.

In all, this volume contains 1037 subject headings as compared with 974 in the first volume, 1758 in the second, 1097 in the third, 872 in the fourth, 908 in the fifth, 1228 in the sixth, and 1322 in the seventh; the total number of rubrics for the eight volumes has therefore been nine thousand one hundred and ninety-six, giving an average of over eleven hundred for each volume.

J. W. BALLANTYNE.

May 30, 1908.

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ENCYCLOPEDIA AND DICTIONARY OF MEDICINE AND SURGERY

Physiology.

PART II.—Nutrition of the Tissues

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SECTION V.—FLUIDS BATHING THE TISSUE

BLOOD AND LYMPH.—The blood carries the necessary nourishment to the tissues and receives their waste products. But it is enclosed in a closed system of vessels, and does not come into direct relationship with the cells. Outside the blood-vessels, and bathing the cells, is the lymph which plays the part of middleman between the blood and the tissues, receiving nourishment from the former for the latter, and passing the waste from the latter into the former.

A. BLOOD

The various physical, chemical, and histological characters of blood must be practically investigated.

I. *General Characters*

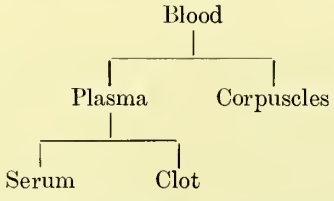
Colour.—Blood changes its colour from purple to cinnabar red on shaking with air, showing that the pigment of the blood may exist in two conditions. *Elements of Blood.*—Microscopic examination shows that blood is composed of a clear fluid (Liquor Sanguinis or plasma) in which float myriads of small disc-like yellowish-red cells (Erythrocytes), and a smaller number of greyish cells (Leucocytes), and certain more minute grey particles (Blood Platelets). The opacity of blood is due to the erythrocytes, and when the pigment is dissolved out of them by water, and they are rendered transparent, the blood as a whole becomes transparent and is said to be "laked." The specific gravity is about 1055. It may be estimated by finding the specific gravity of a sodium sulphate solution in which a drop of blood neither sinks nor floats.

Taste and smell are characteristic, and must be experienced. *Reaction.*—Blood is alkaline, and the degree of alkalinity is very constant in health. It is equivalent to about 0·3 per cent of Na_2CO_3. It is increased during digestion and diminished after muscular exercise.

The cells of the blood constitute about 33 per cent of its weight, and the total solids of the blood are about 20 per cent.

Clotting or Coagulation.—In the course of about three minutes the blood when shed becomes a solid jelly. The process starts from the sides of the vessel, and spreads throughout the blood until, when clotting is complete, the vessel may be inverted without the blood falling out. In a short time, drops of clear fluid appear upon the surface of the clot, and in a few hours these have accumulated to a considerable extent, while the clot has contracted and drawn away from the sides of the vessel, until it finally floats in the clear fluid—the serum. If clotting occurs slowly, as it does in the horse, the erythrocytes subside, leaving a layer of clear plasma above, which, when coagulation takes place, forms a "buffy layer" in the upper part of the clot. Clotting is due to changes in the plasma, since this fluid will coagulate in the absence of corpuscles.

The change may be represented thus:—



The change consists in the formation of a series of fine elastic threads of fibrin throughout the plasma, and if red corpuscles are present they are entangled in the meshes of the network and give the clot its red colour.

These threads may be readily collected in mass upon a stick with which the blood is whipped as it is shed. The red fluid blood which is left, consisting of blood cells and serum, is said to be *defibrinated.*

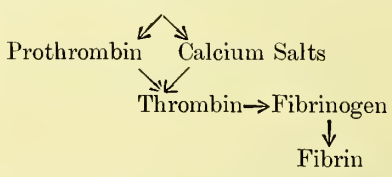
Fibrin is a protein substance. It is slowly dissolved in solutions of neutral salts. It is coagulated by heat, and is precipitated when an excess of a neutral salt is added. It is therefore a globulin.

The plasma before clotting and the serum squeezed out from the clot both contain in the same proportion an albumin (serum albumin) and a globulin, or series of globulins which may be classed together as serum globulin. But the plasma contains a small quantity—about 0·4 per cent—of another globulin (fibrinogen) which coagulates at a low temperature, and which is absent from serum. It is this which undergoes the change from the soluble form to the insoluble form in coagulation. If, by taking advantage of the fact that it is more easily precipitated by sodium chloride than the other proteins, it is separated from them, it may still be made to clot.

The substance which usually causes clotting appears to be an enzyme, which is formed by the action of calcium salts or ions on a proenzyme set free in the blood. The enzyme may be called thrombin, and its precursor prothrombin. Oxalates, when added to blood, precipitate the soluble calcium salts, and prevent the formation of thrombin, and thus prevent coagulation.

It is not yet quite clear what brings about this change of prothrombin into thrombin. Calcium salts or calcium ions and prothrombin may exist together without thrombin being formed, and it has been suggested that another enzyme is required to activate the thrombin—a thrombokinase as it has been called. This may be liberated from the cellular elements of the blood and tissues.

The steps in the process of clotting might therefore be represented as follows:—



Many circumstances influence the rapidity of clotting. Temperature has a marked effect; a low temperature retarding it, a slight rise of temperature above the normal of the particular animal accelerating it. If a trace of a neutral salt be added to blood, coagulation is accelerated; but, if blood be mixed with strong solutions of salt, coagulation is prevented because the formation of thrombin is checked. Calcium salts have a marked and important action, and if they are precipitated by the addition of oxalate of soda, blood will not clot, apparently because thrombin cannot be formed.

The injection into the blood-vessels of a living animal of *commercial peptones*, which chiefly consist of proteoses, or of *hirudin*, an extract *of the head of the medicinal leech*, retards coagulation after the blood is shed. They appear to cause the development in the liver of some body of the nature of an antithrombin which checks coagulation, and if the liver be excluded from the circulation this is not developed.

Why is it that blood does not coagulate in the vessels and does coagulate when shed? Such a general statement is not absolutely correct, for blood may be made to coagulate in the vessels of a living animal in various ways. If inflammation is induced in the course of a vessel, coagulation at once occurs. If the inner coat of a vessel be torn, as by a ligature, or if any roughness occurs on the inner wall of a vessel, coagulation is apt to be set up. Again, various substances injected into the blood stream may cause the blood to coagulate, and thus rapidly kill the animal. Among such substances are extracts of various organs—thymus, testis, and lymph glands—which yield thrombokinase, and snake venom which seems to contain active thrombin. The injection of pure thrombin does not usually cause clotting, because an anti-thrombin is developed.

Nor does blood necessarily coagulate when shed. If it is received into castor oil, or into a vessel anointed with vaseline and filled with paraffin oil, it will remain fluid for a considerable time. Apparently some roughness in the wall of the blood-vessel or of the vessel in which the blood is received is required to start the process, acting as a focus from which it can spread outwards.

The advantages of coagulation of blood are manifest. By means of it wounds in blood-vessels are sealed and hæmorrhage stopped.

Although an important and very prominent change in the blood, clotting is really produced by change in one constituent of the plasma, which is present in very small quantities.

II. *Plasma and Serum*

These may be considered together, since serum is merely plasma *minus* fibrinogen. As serum is so much easier to procure, it is generally employed for analysis.

Both are straw-coloured fluids, the colour being due to a yellow lipochrome. Sometimes they are clear and transparent, but after a fatty diet they become milky. They are alkaline in reaction, and have a specific gravity of about 1025. They contain about 90 per cent of water and 10 per cent of solids. The chief solids are the proteins—serum albumin and serum globulin (with, in the plasma, the addition of fibrinogen). The proportion of the two former proteins to one another varies considerably in different animals, but in the same animal at different times the variations are small. The globulin probably consists of at least two bodies—euglobulin precipitated by weak acid, and pseudoglobulin not so precipitated. The amount of albumin is generally greater when the body is well nourished. In man, they together form about 7 per cent of the serum.

The other organic constituents of the serum are in much smaller amounts, and may be divided into—

1. *Substances to be used by the Tissues.*—Glucose is the most important of these. It occurs only in small amounts—about 1 to 2 per mille. Part of it is free, but part is probably combined in organic combinations such as jecorin. It is probably in larger amount in blood going to muscles than in blood coming from muscles, and this difference seems to be specially well marked when the muscles are active.

Fats occur in very varying amounts, depending upon the amount taken in the food.

2. *Substances given off by the Tissues.*—The chief of these is urea, which occurs constantly in very small amounts in the serum—about ·05 per cent. We shall afterwards see that it is derived from the liver, and that it is excreted in the urine by the kidneys.

Creatin (vol. vii. p. 503), with uric acid (p. 87), and some allied bodies appear to be normally present in traces, and their amount may be increased in diseased conditions.

Of the inorganic constituents of the serum the most abundant is chloride of sodium, but in addition sodium carbonate and alkaline sodium phosphate are also present. Calcium, potassium, and magnesium occur in very small amounts.

III. *Cells of Blood*

1. *Leucocytes—White Cells.*—These are much less numerous than the red cells, and their number varies enormously in normal conditions. On an average there are about 7500 per cubic millimetre.

They are soft, extensile, elastic, and sticky, and each contains a nucleus and a well-developed double centrosome. In size they vary considerably, some being much larger than the red cells, some slightly smaller. The character of the nucleus varies greatly, and from this and from variations in the protoplasm, they may be divided into four classes.

1*st. Lymphocytes.*—Cells with a clear protoplasm and a more or less circular nucleus. Some are very small, while others are larger. They constitute about 20 to 25 per cent of the leucocytes (Fig. 110, *b* and *c*).

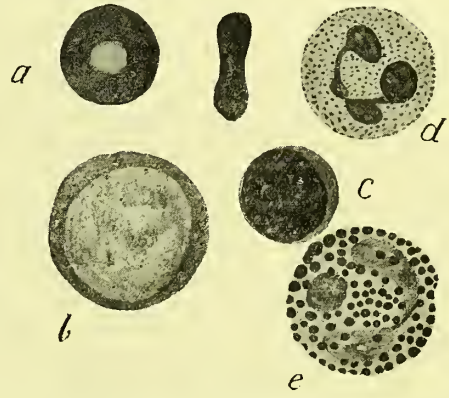


FIG. 110.—Cells of the blood. *a*, Erythrocytes; *b*, large, and *c*, small lymphocyte; *d*, polymorpho-nuclear leucocyte; *e*, eosinophil leucocyte.

2*nd. Polymorpho-nuclear leucocytes*, with a much-distorted and lobated irregular nucleus and a finely granular protoplasm, whose granules stain with acid and neutral stains. These constitute about 70 to 75 per cent of the leucocytes.

3*rd. Eosinophil or oxyphil leucocytes*, with a lobated nucleus like the last, but with large granules in the protoplasm which stain deeply with acid stains. From 1 to 4 per cent of the leucocytes are of this variety.

4*th. Basophil leucocytes*, practically absent from normal blood, with a lobated nucleus and granules in the protoplasm, staining with basic stains.

Myelocytes are large leucocytes with a large circular or oval nucleus and a finely granular protoplasm. They are not normal constituents

of the blood, but appear when the activity of the bone-marrow is increased in certain pathological conditions.

These various forms have certain properties—(*a*) *Amœboid movement.* They can, under suitable conditions, undergo certain changes in shape, as may be readily seen in the blood of the frog, or other cold-blooded animal. The motion may consist simply of the pushing out and withdrawal of one or more processes (pseudopodia), or, after a process is extended, the whole corpuscle may follow it and thus change its place, or the corpuscle may simply retract itself into a spherical mass. As a result of these movements the corpuscles, in certain conditions, creep out of the blood-vessels and wander into the tissues (*Diapedesis*).

(*b*) *Phagocyte Action.*—The finely granular leucocytes and the lymphocytes have further the power of taking foreign matter into their interior, and of thus digesting it. By this devouring action, useless and effete tissues are removed and dead micro-organisms in the body are taken up and got rid of. This scavenger action of the leucocytes is of vast importance in pathology.

Chemistry of Leucocytes.—The nucleus is chiefly made up of nuclein, and in the protoplasm a nucleo-protein, along with two globulins and a small amount of an albumin, are found. Along with these protein substances glycogen and a small amount of fat are present, while the chief inorganic constituents are potassium salts.

2. *Blood Platelets.*—These are small circular or oval discoid bodies, about one-third the diameter of a red blood corpuscle. Some observers have stated that they contain a central nucleus. They are very sticky, and mass together when blood is shed and adhere to a thread passed through blood or to any rough point in the lining of the heart or vessels. They there form clumps, and from these clumps fibrin threads are seen to shoot out. They thus appear to play an active part in clotting. They are present in the blood of mammals only. Their source is not definitely known, but it has been suggested that they are the extruded nuclei of developing erythrocytes.

3. *Erythrocytes—Red Cells.*—All mammals except the camels have circular, biconcave, discoid erythrocytes, which, when the blood is shed, tend to run together like piles of coins. The camels have elliptical biconvex corpuscles. A nucleus is not present in the fully-developed mammalian erythrocyte. In birds, reptiles, amphibia and fishes, the corpuscles are elliptical biconvex bodies, with a well-marked central nucleus. The size of the human erythrocytes is fairly constant—on an average 7·6 micromillimetres in diameter. The number of red cells in health is about 5,000,000 in men and 4,500,000 in women, per cubic millimetre; but in disease it is often decreased.

The number of corpuscles per cubic millimetre is estimated by the Hæmocytometer. This consists of (1) a pipette by which the blood may be diluted to a definite extent with normal salt solution; and (2) a cell of definite depth ruled in squares, each containing above it a definite small volume of blood so that the number of corpuscles in that volume may be counted under the microscope.

The pale yellow colour of the individual corpuscles is due to a pigment held in a fine sponge-like stroma which seems to form a capsule round the cell. This pigment may be dissolved out by various agents, and the action is termed *Hæmolysis.* It may be brought about in different ways:—1*st.* By placing the erythrocytes in a fluid of lower osmotic equivalent, *i.e.* of lower molecular concentration, than the blood plasma and corpuscles. A solution of 0·9 per cent of sodium chloride has the same osmotic equivalent as the plasma and preserves the corpuscles unaltered; in more dilute fluid the corpuscles tend to swell up by endosmosis and the pigment is dissolved out. Erythrocytes may therefore be used as a means of determining the osmotic equivalent—the molecular concentration of a fluid. 2*nd.* By the action of substances which dissolve some constituent of the stroma, *e.g.* salts of the bile acids, chloroform, ether, etc. 3*rd.* By Hæmolysins. The serum of one animal contains a substance, destroyed by heating to 55° C., which is hæmolytic to the blood of animals of other species; *e.g.* the serum of eels' blood contains a powerful hæmolysin for rabbits' erythrocytes, and the serum of the dog a less powerful one. Further, by injecting the blood or the erythrocytes of one species of animal into another species, a hæmolysin is developed which has a specific action on the erythrocytes of the first species.

Chemistry.—The *stroma* of the erythrocytes is made up of a globulin-like substance, in connection with which lecithin and cholesterin occur in considerable quantities. Potassium is the base most abundantly present.

The *pigment* is *hæmoglobin.* It constitutes no less than 90 per cent of the solids of the corpuscles. In many animals, when dissolved from the corpuscles, it crystallises very readily. The crystals prepared from the human blood are rhombic plates. When exposed to air they are of a bright red colour, but if placed in the receiver of an air-pump at the ordinary temperature they become of a purplish tint. The same thing occurs if the hæmoglobin is in solution, or if it is still in the corpuscles. The addition of any reducing agent, such as ammonium sulphide or a ferrous salt, also causes a similar change. This is due to the fact that *hæmoglobin has an affinity for oxygen*, which it takes up from the air, forming a definite compound of a bright red colour in which one molecule of hæmoglobin links with a molecule of oxygen, HbO_2, and is known as *oxyhæmoglobin.*

Hæmoglobin is closely allied to the proteins, but differs from them in containing 0·42 per cent of iron.

When light from the sun is allowed to pass through solutions of blood pigments, certain parts of the solar spectrum are absorbed, and when the spectrum is examined dark bands—the absorption bands—are seen. In a weak solution of oxyhæmoglobin a dark band is seen in the green and another in the yellow part of the spectrum between Fraunhofer's lines D and E, while the violet end of the spectrum is absorbed (Fig. 111). These bands may be broadened or narrowed by strengthening or weakening the solution. When the oxygen is taken away and the purple reduced hæmoglobin is formed, a single broad band between D and E takes the place of the two bands (Fig. 111).

The property of taking oxygen from the air and of again giving it up at a moderate temperature and under a low pressure of oxygen is the great function of the blood pigment in the body. The hæmoglobin plays the part of a middleman between the air and the tissues, taking oxygen from the one and handing it on to the others.

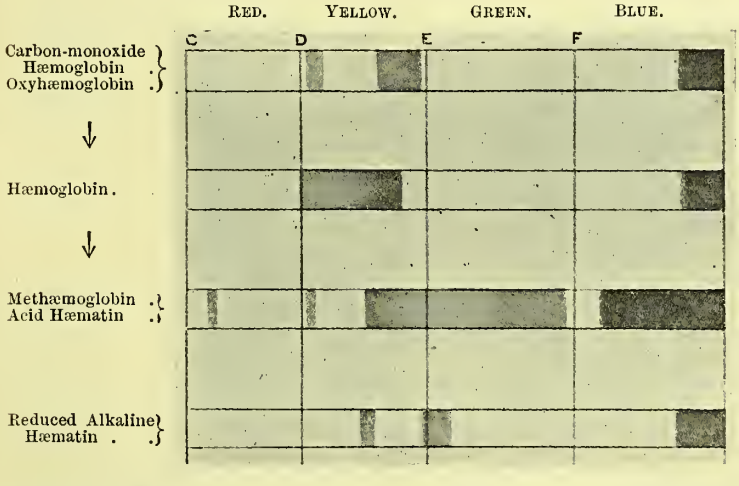


Fig. 111.—Spectra of the more important blood pigments and their more important derivatives. (The spectrum of acid hæmatin is not *identical* with that of methæmoglobin.)

Hæmoglobin constitutes about 13 or 14 per cent of the blood, but in various diseases its amount is decreased. The best method of estimating its amount is by Haldane's Hæmoglobinometer. This consists of two tubes of uniform calibre, one filled with a 1 per cent solution of normal blood saturated with CO, and another in which 20 cmm. of blood to be examined, measured in a pipette, is placed in water, mixed with coal gas to saturate with CO, and then diluted till it has the same tint as the standard tube. The percentage of hæmoglobin in terms of the normal is indicated by the mark on the tube at which the fluid stands.

Methæmoglobin.—Hæmoglobin forms another compound with oxygen—methæmoglobin. The amount of oxygen is the same, but methæmoglobin must be acted on by the strongest reducing agents before it will part with its oxygen. When, therefore, this pigment is formed in the body, the tissues die from want of oxygen. It may be produced by the action of various substances on oxyhæmoglobin. Among these are ferricyanides, nitrites, and permanganates. It crystallises in the same form as oxyhæmoglobin, but has a chocolate-brown colour. Its spectrum is also different from hæmoglobin or oxyhæmoglobin, showing a narrow sharp band in the red part of the spectrum, with two or more bands in other parts according to the reaction of the solution in which it is dissolved (Fig. 111). It is of importance since it occurs in the urine in some pathological conditions. In all probability the molecule of oxyhæmoglobin has the formula—$Hb\begin{matrix}O\\|\\O\end{matrix}$, while in methæmoglobin the atoms are arranged $Hb\overset{O}{\underset{O}{\diagdown\!\!\diagup}}$.

Hæmoglobin also combines with certain other gases. Among these is carbon monoxide. For this gas hæmoglobin has a greater affinity than for oxygen, so that when carbon monoxide hæmoglobin is once formed in the body, the blood has little power of taking up oxygen, and the animal dies. This gas is evolved freely in the fumes from burning charcoal, is present in coal gas, and is found in the air of coal-mines after explosions. Carbon monoxide hæmoglobin forms crystals like oxyhæmoglobin, and has a bright *pinkish*-red colour, without the yellow tinge of oxyhæmoglobin. Since after death it does not give up its carbon monoxide and become changed to purple hæmoglobin, the bodies of those poisoned with the gas maintain the florid colour of life. Its spectrum is very like that of oxyhæmoglobin, the bands being slightly more to the blue end of the spectrum (Fig. 111).

It may be at once distinguished by the fact that when gently warmed with ammonium sulphide it does not yield reduced hæmoglobin.

Decomposition of Hæmoglobin.—Hæmoglobin is a somewhat unstable body, and, in the presence of acids and alkalies, splits up into about 96 per cent of a colourless protein *globin* belonging to the globulin group, and about 4 per cent of a substance of a brownish colour called *hæmatin.*

The spectrum and properties of this substance

are different in acid and alkaline media. In acid media it has a spectrum closely resembling methæmoglobin, but it can at once be distinguished by the fact that it is not changed by reducing agents. In medicine it is sometimes important to distinguish between these pigments since both may appear in the urine. Hæmatin in alkaline solution can take up and give off oxygen in the same way as hæmoglobin does. Reduced alkaline hæmatin or *hæmochromogen* has a very definite spectrum (Fig. 111), and its preparation affords a ready means of detecting old blood stains. Hæmatin contains the iron of the hæmoglobin, and it is this pigmented iron-containing part of the molecule which has the affinity for oxygen. Apparently it is the presence of iron which gives it this property, because, if the iron be removed by means of sulphuric acid, a purple-coloured substance, *iron-free hæmatin—hæmatoporphyrin*—is formed, which has no affinity for oxygen. This pigment occurs in the urine in some pathological conditions.

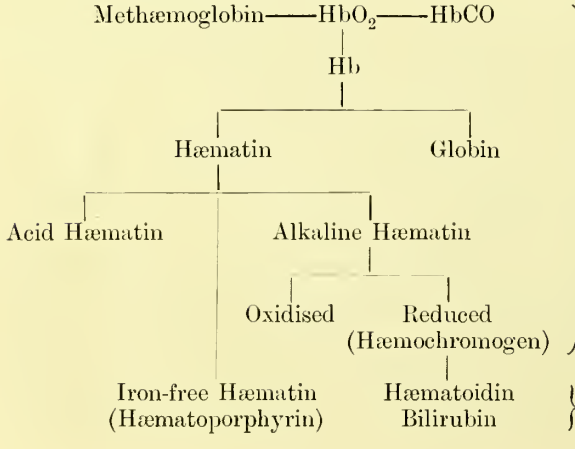
One point of great interest in the chemistry of hæmatin and its derivatives is that they, like the green chlorophyl of plants, yield upon decomposition bodies belonging to the pyrrhol group.

In the liver, hæmoglobin is broken down to form *bilirubin* and the other bile pigments. These are iron-free, and, like iron-free hæmatin, do not take up and give off oxygen. But not only is this iron-free pigment formed from hæmoglobin in the liver, but the cells of any part of the body have the faculty of changing hæmoglobin in blood extravasations into a pigment known as *hæmatoidin*, which is really the same as bilirubin.

Hæmin—the hydrochloride of hæmatin—is formed when blood is heated with sodium chloride and glacial acetic acid. It crystallises in small steel-black rhombic crystals, and its formation is sometimes used as a test of blood stains.

The following table shows the relationship of these pigments to one another :—

Relationship of Hb and its Derivatives



IV. *Gases of the Blood*

The muscles and other active tissues are constantly consuming oxygen and constantly giving off carbon dioxide. The oxygen must be brought to the tissues by the blood, and the carbon dioxide carried away by the same medium.

Various methods of carrying out the examination of the gases of the blood have been devised, and many different gas-pumps have been invented in which the gases may be collected in the Torricellian vacuum over mercury. Haldane and Barcroft have devised a convenient method, which depends upon the fact that the oxygen can be driven off from blood treated with dilute ammonia by the addition of potassium ferricyanide, and that the carbon dioxide is liberated by adding an acid. The amount of gas is estimated by measuring the increased pressure in the tube in which the gas has been given off.

About 60 c.c. of gas measured at 0° C. and 760 mm. pressure can be extracted from 100 c.c. of blood. The proportion of the gases varies in arterial and in venous blood.

Amount of Gases per Hundred Volumes of Blood

	Arterial Blood.	Venous Blood.
Oxygen . .	20	12
Carbon dioxide .	40	46

There are two ways in which gases may be held in such a fluid as the blood—

1*st*. In simple solution.
2*nd*. In chemical combination.

Oxygen.—At the temperature of the body the blood can hold in solution less than 1 per cent of oxygen. Now the amount of oxygen actually present is about 20 per cent; so that by far the greater quantity of the gas is not in solution. We have already seen that it is in loose chemical union with hæmoglobin.

Carbon Dioxide.—In the animal body the blood can dissolve about $2\frac{1}{2}$ per cent of carbon dioxide. But it may contain as much as 46 per cent, and this is uniformly distributed between plasma and corpuscles. Hence the greater part of the gas must be in chemical combination. Analysis of the ash of the plasma shows that the sodium is more than sufficient to combine with the chlorine and phosphoric acid, and is thus available to take up carbon dioxide, as the carbonate Na_2CO_3 and the bicarbonate $NaHCO_3$. Sodium carbonate and basic

sodium phosphate are therefore present together in the plasma.

If carbon dioxide is passed into a solution of sodium phosphate it appropriates a certain amount of the sodium, changing Na_2PO_4 to $NaHPO_4$. This is what happens in the tissues where CO_2 is abundant. In the lungs, where the blood is

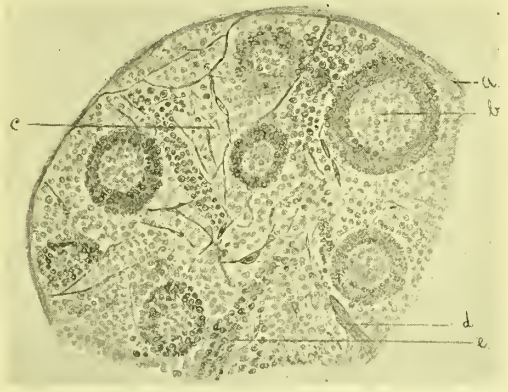


FIG. 112.—Section of a lymph gland. *a*, Capsule; *b*, germ centres of cortex; *c*, sinuses; *d*, trabecula; *e*, germ centres of medulla.

exposed to an atmosphere poor in CO_2, the P_2O_5 again seizes on the Na, turning out the CO_2.

But the proteins of the blood also act in the same way as weak acids, being turned out of their combination with bases by the mass action of CO_2, and thus acting as carriers of CO_2.

In the corpuscles the hæmoglobin acts as an acid. When the amount of CO_2 is great, hæmoglobin is turned out of its combination with bases. But when the pressure of CO_2 is low the hæmoglobin turns it out. For this reason it is possible to remove all the CO_2 from whole blood in an air-pump, but not possible to remove it from blood plasma. In fact, the carriage of carbon dioxide and its excretion are mainly the result of a struggle between that gas on the one hand and the proteins and phosphoric acid on the other, for the bases of the plasma.

Nitrogen.—The amount of nitrogen in the blood is not in excess of what can be held in solution, and we may therefore infer that it is simply dissolved in the blood plasma.

V. *Source of the Blood Constituents*

A. *Of the Plasma.*—The water of the blood is derived almost entirely from the water ingested.

The source of the proteins has not been fully investigated. Undoubtedly they are partly derived, somewhat indirectly as we shall afterwards see, from the proteins of the food. Very probably, too, they are in part derived from the tissues. But the significance of the two proteins, albumin and globulin, and of their variations has not yet been elucidated.

The glucose is derived from the carbohydrates and possibly from the proteins of the food, and during starvation it is constantly produced in the liver and poured into the blood (p. 74).

The fats are derived from the fats and carbohydrates, and possibly from the proteins of the food.

The urea and other waste constituents are derived from the various tissues.

B. *Of the Cells.*—I. *Leucocytes.*—In the embryo these are first developed from the mesoblast cells generally. In extra-uterine life they are formed in the lymph tissue and in the red marrow of bone.

1. *Lymph tissue* (*see* vol. vii. p. 499) is very widely distributed in the body, occurring either in patches of varying shape and size, or as regular organs, the lymphatic glands (Fig. 112). These are placed on the course of lymphatic vessels, and consist of a sponge-work of fibrous tissue, in the interstices of which are set the patches of lymph tissue or *germ centres*, each surrounded by a more open network, the sinus, through which the lymph flows, carrying away the lymphocytes, which are the characteristic elements produced, from the germ centres. Round some of the lymphatic glands of certain animals large blood spaces or sinuses are seen, and these glands are called hæmolymph glands (Fig. 114). They are intermediate between lymphatic glands and the spleen. When erythrocytes are destroyed by hæmolytic agents, the pigment and the iron derived from the hæmoglobin are often found abundantly in the cells in the sinuses of lymph and hæmolymph glands.

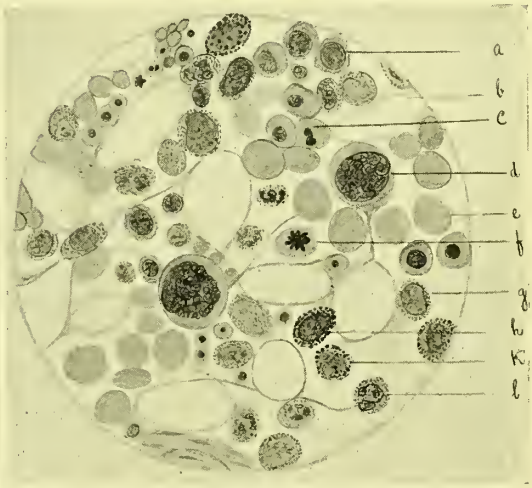


FIG. 113.—Section of red marrow of bone. *a*, Lymphocyte; *b*, fat cell; *c*, erythroblast; *d*, giant cell; *e*, erythrocyte; *f*, erythroblast in mitosis; *g*, neutrophil myelocyte; *h*, eosinophil myelocyte; *k*, eosinophil leucocyte; *l*, polymorphonuclear leucocyte.

2. *Bone Marrow.*—The structure of bone marrow is considered below, but it may be stated here that young leucocytes or leucoblasts, in the condition of mitosis, are abundant, and that they pass away in the blood stream. They are of all varieties. In certain pathological conditions the formation of these cells is increased and a leucocytosis results (Fig. 113).

II. *Erythrocytes.*—In the embryo these cells

seem to be formed by a process of budding from the mesoblast cells, which become vacuolated to form the primitive blood-vessels. Later they develop in the liver and spleen, but after birth they are formed in the red marrow of bone (Fig. 113). Marrow consists of a fine fibrous tissue with large blood capillaries or sinuses running in it. In the fibrous tissues are numerous *fat cells* (clear spaces *b* in Fig. 113) and generally a considerable number of multinucleated *giant cells* or myelocytes (*d*). In addition to these are the young leucocytes, *leucoblasts* (*a.g.h.*), and lastly young nucleated red cells, the *erythroblasts* (*c.f.*). After hæmorrhage, the formation of these becomes unusually active, and may implicate parts of the marrow not generally concerned in the process, and hence the red marrow may spread from the ends of the long bones, where it is usually situated, towards the middle of the shaft. The nuclei of the erythroblasts atrophy or are shed, and the cells escape into the blood stream. The red marrow has the power of retaining the iron of disintegrated erythrocytes, which are often found enclosed in large modified leucocytes or phagocytes. The iron is often very abundant after a destruction of erythrocytes.

VI. *Total Amount of Blood in the Body*

This was formerly determined by bleeding an animal, measuring the amount of blood shed, and determining the amount of hæmoglobin contained in it; then washing out the blood-vessels, and after measuring the amount of fluid used, determining the amount of hæmoglobin in it to ascertain the amount of blood it represented. By this method the amount of blood was found to be about $\frac{1}{13}$ of the body weight.

Haldane and Lorrain Smith have devised a method which can be applied to the living animal. It depends upon the fact that, after an animal or person has inhaled carbon monoxide, it is possible to determine to what proportion the gas has replaced oxygen in the oxyhæmoglobin. If then an individual breathes a given volume of carbon monoxide, and if a measured specimen of blood is found to contain a definite percentage of the gas, the rest of the gas must be equally distributed through the blood, and thus the amount of blood may be deduced.

By this method they conclude that the blood is about $\frac{1}{20}$ of the weight of the body in the human subject.

VII. *Distribution of the Blood*

Roughly speaking, the blood is distributed somewhat as follows:—

Heart, lungs, large vessels . .	$\frac{1}{4}$
Muscles	$\frac{1}{4}$
Liver	$\frac{1}{4}$
Other organs	$\frac{1}{4}$

VIII. *Fate of the Blood Constituents*

The water of the blood, constantly renewed from outside, is constantly got rid of by the kidneys, skin, lungs, and bowels.

About the fate of the proteins we know nothing. They are probably used in the construction of the tissues, but experimental evidence of this is wanting.

The glucose and fat are undoubtedly used up in the tissues.

The urea and waste products are excreted by the kidneys.

The fate of the salts is not fully worked out. The chlorides are partly excreted by the kidneys and are partly split up to form the hydrochloric acid required for stomach digestion. The phosphates and sulphates are excreted in the urine, but whether they are also used in the tissues is not known.

The leucocytes break down in the body—but when and how we do not know. We shall afterwards find that they are greatly increased in number after a meal of proteins, and, since the increase is transitory, lasting only for a few hours, they are probably rapidly broken down, possibly to feed the tissues. It would thus seem that a leucocyte may live for only a short time in the blood.

The erythrocytes also break down. How long they live is not known. It is found, after injecting blood, that the original number of corpuscles is not reached for about a fortnight, and hence it has been concluded that the corpuscles live for that period. The experiment, however, is far from conclusive, and must be accepted with reservation.

Organs connected with Hæmolysis. — The process of breaking down of old erythrocytes and eliminating their pigment is often called the process of hæmolysis. Certain organs appear to be specially connected with it, but the precise part played by each of them is not very clearly understood.

That the liver acts in this way is indicated, first, by the fact that the blood passing from the organ during digestion contains fewer erythrocytes than the blood going to it; second, by the formation in the liver cells of bile pigments, which are derivatives of hæmoglobin; third, by the presence of pigment and of iron in simple combinations in the liver cells under certain conditions. It is possible that the reabsorbed salts of the bile acids in the portal blood dissolve the pigment out of the old erythrocytes, and that the liver cells may then act upon the liberated pigment. Under ordinary conditions the liver does not store much iron.

The spleen is generally said to have a similar action. This organ is composed of a fibrous capsule containing non-striped muscle and a sponge-work of fibrous and muscular trabeculæ,

in the interstices of which is the spleen pulp. The branches of the splenic artery run in the trabeculæ, and twigs pass out from these trabeculæ, and are covered with masses of lymph tissue forming the Malpighian corpuscles. Beyond these, the vessels open into a series of complex sinuses lined by endothelial cells of large size, from which the blood is collected into channels, the venous sinuses, which carry it back to branches of the splenic vein in the trabeculæ. The pulp is thus comparable with the blood sinuses of the hæmolymph glands, and the spleen may be considered as being a still further development of the hæmolymph gland (Fig. 114).

So far, no decrease in the number of erythrocytes in the blood leaving the spleen has been recorded. In the cells of the spleen pulp, and chiefly in the endothelial cells, yellow pigment and simple iron compounds are frequently found, indicating that hæmoglobin is being broken down. But the idea that the spleen plays an important part in the actual destruction of erythrocytes seems to be negatived by the fact that, when blood is injected, the cells are broken down no faster in an animal with the spleen intact than in an animal from which the spleen has been removed. While the spleen appears to have no action in killing and destroying erythrocytes, its cells, like those of the sinuses of the lymph glands and hæmolymph glands, have the power of taking up dead and disintegrating erythrocytes and storing the iron for future use in the body. These organs may, in fact, be regarded as the graves of the dead erythrocytes.

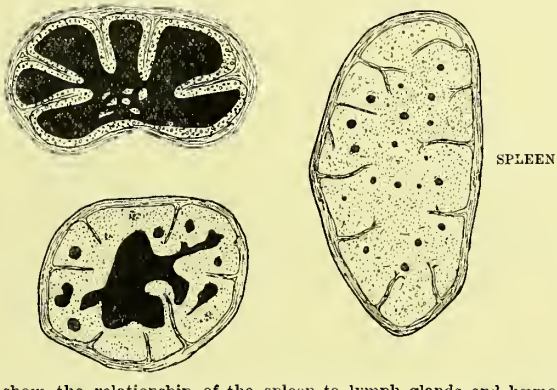


FIG. 114.—To show the relationship of the spleen to lymph glands and hæmolymph glands. The black indicates lymphoid tissue; the coarsely spotted part, lymph sinuses, and the finely dotted part, blood sinuses. (Lewis.)

The non-striped muscle in the framework of the spleen undergoes rhythmic contraction and relaxation, and the organ thus contracts and expands at regular intervals of about a minute.

These movements may be recorded by enclosing the organ in an oncometer, a closed capsule connected with some form of recording apparatus.

The movements are controlled by fibres leaving the spinal cord chiefly in the 6th, 7th, and 8th dorsal nerves of both sides. Strong stimulation of these causes contraction.

B. Lymph

(1) *Characters of Lymph.*—The lymph is the fluid which plays the part of middleman between the blood and the tissues. It fills all the spaces in the tissues and bathes the individual cell elements. These spaces in the tissues open into vessels—the lymph vessels—in which the lymph flows and is conducted through lymph glands and back to the blood through the thoracic duct (*see* Fig. 115, p. 10).

Lymph varies in character according to the situation from which it is taken and according to the condition of the animal.

Lymph taken from the lymph spaces—*e.g.* the pericardium, pleura, or peritoneum—is a clear straw-coloured fluid. It has little or no tendency to coagulate. Microscopic examination shows that it contains few or no cells—any cells which may exist being lymphocytes. In reaction it is alkaline. The specific gravity varies according to its source, being lowest when from the limbs and highest when from the liver.

Apparently the cause of the non-coagulation of such lymph is the absence of cells from which thrombokinase may be set free. If blood or white corpuscles be added to it, a loose coagulum forms.

If the lymph be taken from lymphatic vessels after these have passed through lymphatic glands, it is found to contain a number of lymphocytes, and it coagulates readily.

Chemically, lymph resembles blood plasma in which the proteins are in smaller amount, but the inorganic salts in the same proportion as in the blood. The amount of solids varies in lymph from different organs.

Lymph of	Proteins.
Limbs . . .	2-3 per cent
Intestines . . .	4-6 ,,
Liver . . .	6-8 ,,

In the lymphatics coming from the alimentary canal during starvation, the lymph has the characters above described. But after a meal it has a milky appearance and is called chyle. This milky appearance is due to the presence of fats in a very fine state of division, forming what is called the molecular basis of the chyle.

Lymph in various diseases tends to accumulate as serous effusions in the large lymph spaces—*e.g.* the pleura, peritoneum, pericardium—and these effusions behave differently as regards coagulation. The following table helps

to explain this (S.A. is Serum Albumin, S.G. Serum Globulin):—

Coagulability of Lymph, Serum, and Effusions.

Plasma and Lymph.	Serous Effusion.			Serum.
	Coag.	Coag. with Thrombin.	Uncoag.	Uncoag.
S. A.	S. A.	S. A.	S. A.	S. A.
S. G.	S. G.	S. G.	S. G.	S. G.
Fibrinogen.	Fibrinogen.	Fibrinogen.	...	...
Thrombin.	Thrombin.	...	...	Thrombin.

2. *Formation of Lymph.*—Lymph is derived partly from the blood and partly from the tissues. The formation of lymph from the blood depends upon the permeability of the walls of the capillaries and the pressure of blood in the blood-vessels. Thus, although the pressure in the blood-vessels of the limbs is much higher than the pressure in the vessels of the liver, hardly any lymph is usually produced in the former, while very large quantities are produced in the latter—apparently because of the small permeability of the limb capillaries and the great permeability of the hepatic capillaries. The permeability may be increased by anything which injures the capillary wall. Thus the injection of hot water or of proteoses at once leads to an increased flow of lymph.

While the permeability of the vessel wall is the most important factor controlling lymph formation, any increase of the intravascular pressure of a region increases the flow of lymph, and for this reason any obstruction to the free flow of blood from a part leads to increased lymph production from that area.

That lymph is also formed from the tissues is indicated by the fact that the injection of substances of high osmotic equivalent into the blood—such as sugar or sodium sulphate—leads, by a process of osmosis, to a flow of fluid into the blood, so that it becomes diluted, and also to an increased formation of and flow of lymph, and this increase of water in both can be explained only by its withdrawal from the tissues.

The Circulatory System

I. *General Arrangement*

The arrangement by which the blood and lymph are distributed to the tissues may be compared to a great irrigation system.

It consists of a central force-pump — the *systemic heart* (Fig. 115, *S.H.*)—from which pass a series of conducting tubes—the *arteries* —leading off to every part of the body, and ending in innumerable fine irrigation channels —the *capillaries* (*Cap.*)—in the substance of the tissues. From these, a considerable proportion of the blood constituents is passed into the spaces between the cells as lymph. From these spaces the fluid is either passed back into the capillaries, or is conducted away in a series of *lymph vessels*, which carry it through *lymph glands* (*Ly.*), from which it gains certain necessary constituents, and finally bring it back to the central pump.

The fluid, which has not passed out of the capillaries into the tissues, has been deprived of many of its constituents, and this withdrawal of nutrient material by the tissues is made good by a certain quantity of the blood being sent through the walls of the stomach and intestine (*Al.C.*), in which the nutrient material of the food is taken up and added to the blood returning to the heart. At the same time, the waste materials added to the blood by the tissues are partly got rid of by a certain quantity of the blood being sent through the liver and kidneys (*Liv.* and *Kid.*).

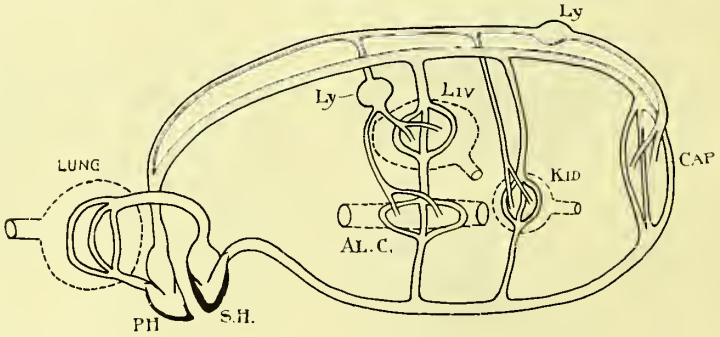


Fig. 115.—Scheme of the circulation. *S.H.*, Systemic heart sending blood to the capillaries in the tissues, *Cap.* The blood brought back by veins and the exuded lymph by lymphatics, *Ly.*, passing through glands; blood sent to the alimentary canal, *Al.C.*, and from that to the liver, *Liv.*; blood also sent to the kidneys, *Kid.*; the blood before again being sent to the body is passed through the lungs by the pulmonic heart, *P.H.*

The blood is then poured back, not at once into the great pump which sends it through the body, but into a subsidiary pump—the *pulmonic heart* (*P.H.*)—by which it is pumped through the lungs, there to obtain a fresh supply of oxygen, and to get rid of the carbon dioxide excreted into it by the tissues. Finally the blood, with its fresh supply of oxygen from the lungs, and of nourishing substances from the alimentary canal, is poured into the great systemic pump—the left side of the heart—again to be distributed to the tissues.

Thus the circulation is arranged so that the blood, exhausted of its nourishing material by the tissues, is replenished in the body before being again supplied to the tissues.

The sectional area of this irrigation system varies enormously. The aorta leaving the heart has a comparatively small channel. If all the arteries of the size of the radial were cut across

and put together, their sectional area would be many times the sectional area of the aorta. And, if all the capillary vessels were cut across and placed together, the sectional area would be about 700 times that of the aorta.

From the capillaries, the sectional area of the veins and lymphatics steadily diminishes as the smaller branches join with one another to form the larger veins and lymphatics; but, even at the entrance to the heart, the sectional area of the returning tubes, the veins, is about twice as great as that of the aorta (Fig. 140, p. 36).

The circulatory system may thus be compared to a stream which flows from a narrow deep channel, the aorta, into a gradually broadening bed, the greatest breadth of the channel being reached in the capillaries. From this point the channel gradually narrows until the heart is reached.

Hence the blood stream is very rapid in the arteries where the channel is narrow, and very sluggish in the capillaries where the channel is wide, so that in them plenty of time is allowed for exchanges between the blood and the tissues.

II. *The Central Pump—The Heart*

A. Structure.—A very simple form of heart exists in the ascidians. At one point on a large vessel there is a thickening in the wall composed of non-striped muscular fibres. A contraction is seen to pass from one end of this to the other at frequent regular intervals, thus forcing the fluid through the vessels. The embryonic heart in man has a similar structure.

In the snail and cuttle-fish, in addition to the contracting muscular thickening, there is also a thin-walled receiving chamber into which the blood flows before it is expelled onwards. The heart is thus composed of two chambers—

1*st.* A receiving chamber—the auricle.

2*nd.* An expelling chamber—the ventricle.

In fish the heart has a similar structure. But in lung-bearing animals a more complex arrangement is required, and a double heart is found: one concerned with the propulsion of blood to the system generally, and hence called the systemic heart; one propelling blood to the lungs, and hence called the pulmonic heart. In mammals, the former chamber is on the left side, the latter on the right. Each consists of a receiving and expelling chamber—an auricle and a ventricle.

The walls of these chambers are essentially muscular; but this muscular layer, or myocardium, lies between two fibrous layers, the pericardium and the endocardium.

The musculature of the auricles is separate from that of the ventricles, but some fibres more like ordinary visceral fibres than cardiac muscle extend from one to the other. This band of His' plays a most important part in conducting contraction started in the auricles to the ventricles. If the heart be boiled, the auricles, the aorta, and the pulmonary artery may be separated from the ventricles. This is because boiling converts fibrous tissue to gelatine and dissolves it, and it is by white fibrous tissue that the auricles and great arteries are attached to the ventricles. This tissue is arranged in three rings: one encircling the opening between the right auricle and ventricle, and crescentic in shape; one, more circular in shape, encircling in common the left auriculo-ventricular and the aortic orifice; and one encircling the pulmonary opening. The auricles are attached to the auriculo-ventricular rings above, the ventricles are attached below, while the valves of the heart are also connected with them.

The muscular fibres of the auricles are arranged in two badly-defined layers—

1*st.* An outer layer running horizontally round both auricles.

2*nd.* An inner layer arching over each auricle, and connected with the auriculo-ventricular rings.

Contraction of the first layer diminishes the capacity of the auricles from side to side. Contraction of the second pulls the auricles downwards towards the ventricles, and thus diminishes their capacity from above downwards.

The peculiar striped muscle fibres of the auricular wall extend for some distance along the great veins which open into these chambers.

The left ventricle forms the cylindrical core to the heart, and the right ventricle is attached along one side of it. The septum between the ventricles is essentially the right wall of the left

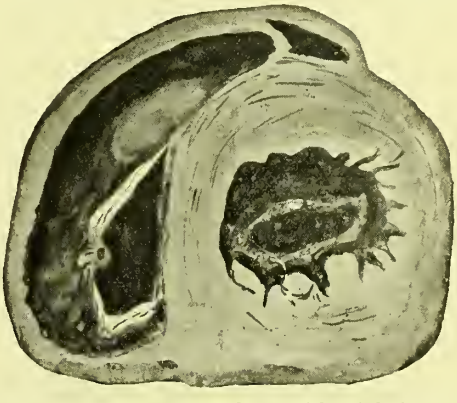


Fig. 116.—Cross section through the ventricles of the heart looking towards auricles, to show the right ventricle placed on the central core of the left ventricle. The cusps of the auriculo-ventricular valves are also shown.

ventricle, and it bulges into the right ventricle with a double convexity from above downwards and from before backwards (Fig. 116).

The muscle fibres of the ventricles are arranged essentially in three layers—

1*st.* The outmost layer takes origin from the auriculo-ventricular rings, and passes downwards and to the left till it reaches the apex of the heart. Here it turns inwards, forming a sort of vortex, and becomes continuous with the inmost layer.

2*nd*. The middle layer is composed of fibres running horizontally round each ventricle. It is the thickest layer of the heart, and in contracting it pulls the walls of the ventricles towards the septum ventriculi.

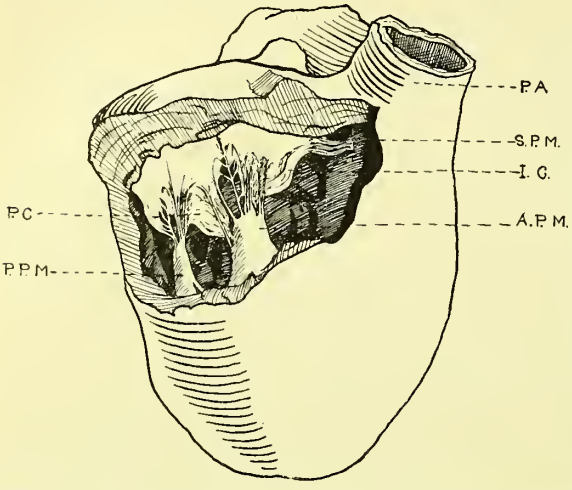


FIG. 117.—The right ventricle and tricuspid valve to show the relationship of the papillary muscles and chordæ tendineæ to the cusps of the valve. (*See* text.)

3*rd*. The inmost layer is continuous with the outmost layer, as it turns in at the apex. It may be considered as composed of two parts—

(*a*) A layer of fibres running longitudinally along the inside of each ventricle from the apex upwards to the auriculo-ventricular ring. These fibres are raised into fleshy columns, the columnæ carneæ.

(*b*) A set of fibres, constituting the papillary muscles (Fig. 117, *P.M.*), which, taking origin from the apical part of the ventricles, extend freely upwards to terminate in a series of tendinous cords (the chordæ tendineæ), which are inserted partly into the auriculo-ventricular valves, presently to be described, and partly into the auriculo-ventricular rings. The papillary muscles are merely specially modified columnæ carneæ. In many cases, actual muscular processes extend from the apex of the papillary muscles to the aurico-ventricular ring.

In the *left ventricle* there are two papillary muscles, or groups of papillary muscles—one in connection with the anterior wall of the ventricle, and one in connection with the posterior wall.

In the *right ventricle* there are—1*st*. One or more small horizontally running papillary muscles just under the pulmonary orifice, their apices pointing backwards (Fig. 117, *S.P.M.*).

2*nd*. A large papillary muscle taking origin from the mass of fleshy columns at the apex of the ventricle (*A.P.M.*).

3*rd*. One or more papillary muscles of varying size arising from the posterior part of the apical portion of the ventricle (*P.P.M.*).

4*th*. A number of small septal papillary muscles arising from the septum.

The distribution of the chordæ from these muscles will be considered in connection with the auriculo-ventricular valves.

In contraction, the outmost and inmost layers of the ventricles tend to approximate the apex to the base of the ventricles, but this is resisted by the contracting middle layer. The apex tends to be tilted towards the right, the papillary muscles shorten; the columnæ carneæ by their shortening and thickening encroach upon the ventricular cavity, and help to abolish it, while the auriculo-ventricular rings are drawn downwards and inwards towards the septum.

The endocardium forms a continuous fibrous layer, lined by endothelium, extending from the vessels over the inner aspect of auricles and ventricles. At certain points flaps of this endocardium are developed to form the valves of the heart.

In the heart, valves are situated at the entrance to and at the exit from the expelling cavities. There is thus on each side of the heart a valve between the auricles and ventricles, and a valve between the ventricles and the great arteries.

Auriculo-ventricular Valves. — On each side of the heart the auriculo-ventricular valve is formed by flaps of endocardium, which hang downwards from the auriculo-ventricular ring like a funnel into the ventricular cavity, and which are attached to the apices of the papillary muscles by the chordæ tendineæ (Figs. 117 and 118).

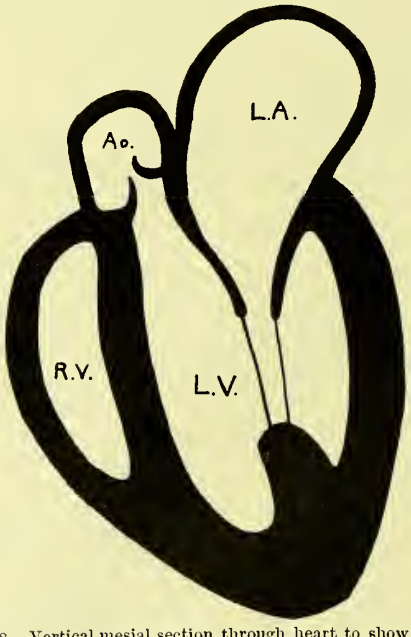


FIG. 118.—Vertical mesial section through heart to show aortic and mitral valves. *R.V.*, Right ventricle; *L.V.*, left ventricle with papillary muscle; *L.A.*, left auricle; *Ao.*, aorta with anterior cusp on top of septum.

On the left side of the heart there are two

main cusps, forming the mitral valves (Fig. 118)—

1*st*. An anterior or right cusp, which takes origin from, and is continuous with, the right posterior wall of the aorta. It hangs down into the ventricle between the aortic and auriculo-ventricular orifices, thus dividing the ventricle into two parts, an aortic and an auricular part. This cusp is very strong, and in many animals bone is developed in it towards its base. It is composed of dense fibrous tissue, is smooth on both sides, and the chordæ are inserted chiefly along its edges.

2*nd*. The posterior or left cusp takes origin from the back part of the auriculo-ventricular ring, and hangs in the ventricle in its relaxed state against the posterior and left wall. It is smaller and less strongly made than the anterior cusp. The chordæ tendineæ are not only inserted into its edge, but run up along its posterior aspect to be inserted into the auriculo-ventricular ring, and they thus give the posterior aspect of the cusp a rough ridged appearance.

When the papillary muscles contract, the cusps are drawn together. The edge of each cusp thins out to form a delicate border, which, when the cusps are approximated, completely seals the aperture.

On the right side of the heart the auriculo-ventricular orifice is separate from the pulmonary opening, and the three cusps of the tricuspid valve are developed in connection with the crescentic opening from the auricle (Fig. 116). One rises from the ring above the septum, and hangs down into the ventricle upon the surface of the septum. This cusp is small, thin, and delicate. It is attached by its lower border to the septal papillary muscles. The chief or infundibular cusp (Fig. 117, *I.C.*) rises from the front part of the ring between the pulmonary infundibulum and the auriculo-ventricular opening. It is connected by its anterior border with the horizontal fibres from the superior papillary muscles, and by its lower and inferior border with the chordæ from the anterior papillary muscle. When these two sets of papillary muscles contract, this cusp is drawn flat against the bulging septum.

The posterior cusp (*P.C.*) takes origin from the posterior and outer part of the ring, and hangs down into the posterior part of the ventricle. It is connected by its anterior margin with the anterior papillary muscle and by its posterior margin with the posterior papillary muscle. Contraction of these muscles therefore approximate its anterior edge to the infundibular cusp, its posterior edge to the septal cusp, and pull it towards the septum.

In both the infundibular and posterior cusps many of the chordæ pass up to be inserted into the auriculo-ventricular ring.

Semilunar Valves. — The valves, situated at the opening of the ventricles into the great arteries, are also formed as special developments of the endocardium.

Each is composed of three half-moon-shaped membranous pouches attached along their curved margin to the walls of the artery and upper part of the ventricle, and with their concavities directed away from the ventricle. In the centre of the free margin is a fibrous thickened nodule, the corpus Arantii, from which a very thin piece of membrane, the lunule, extends to the attached margin of the edges. A pouch, the sinus of Valsalva, lies behind each cusp.

The arrangements of these various cusps is of importance in connection with their action (Fig. 118).

Aortic Valve.—The anterior cusp is largest, and lies somewhat deeper in the heart than the others. At each side it is attached to the aortic wall, but below it is attached to the upper part of the septum ventriculi, so that the base of the sinus of Valsalva is formed by the upper part of the septum. At a somewhat higher level is a cusp which is partly attached to the upper part of the septum, partly to the posterior wall of the aorta, where this becomes continuous with the anterior cusp of the mitral. The third cusp is still higher, and is attached to the aortic wall, where it becomes continuous with the anterior cusp of the mitral.

Pulmonary Valve. — The posterior cusp is mounted on the top of the septum ventriculi, and is at a somewhat lower level than the other two.

Thus, in each valve, the cusp placed lowest is mounted on a muscular cushion, the use of which will afterwards be considered.

Attachments and Relations of the Heart (Fig. 119).—The heart is attached, by the great vessels coming from it, to the posterior wall of the chest at the level of the fifth to the eighth dorsal vertebræ. Its plane of attachment faces forwards and downwards. From this the heart projects into the chest as a conical mass downwards, forwards, and to the left. It does not lie at right angles to its plane of attachment, but, when not contracting, it is limp and hangs down, as shown in the diagram (continuous line). Nor can it assume a position at right angles to its plane of attachment (dotted line), because the front part of the heart lies against the anterior chest wall over an area (the præcordium) bounded to the right by the midsternal line, above by the fourth left rib, below by the seventh left rib, and to the left by a vertical line inside the nipple line.



FIG. 119.—Mesial section through the thorax to show the attachment and relations of the heart.

From the oblique position of the heart, the right auricle and ventricle are directed forwards, and it is a portion of the right ventricle which lies in relationship with the chest wall. The rest of the organ is covered by the lungs. The only parts of the left side which can be seen from the front are the tip of the left auricular appendix, and a narrow strip of the left ventricle.

Below and behind the heart lies upon the central tendon of the diaphragm, to which the pericardium is attached.

All round it are the lungs completely filling up the rest of the thorax.

The heart is enclosed in a strong fibrous bag, the pericardium, which supports it and prevents over-distension. When fluid accumulates in this bag, the auricles are pressed upon and the flow of blood into them is impeded.

B. PHYSIOLOGY OF THE HEART.—*The Cardiac Cycle.*—Each part of the heart undergoes contractions and relaxations at regular rhythmical intervals, *and the sequence of events from the occurrences of any one event to its recurrence constitutes the cardiac cycle.*

A. *Frog.*—In the frog a contraction, starting from the openings of the veins, suddenly involves the sinus venosus, causing it to become smaller and paler. This contraction is rapid and of short duration, and is followed by a relaxation, the cavity again regaining its former size and colour. As this relaxation begins, the two auricles are suddenly contracted and pulled downwards towards the ventricle, at the same time becoming paler, while the ventricle becomes more distended and of a deeper red. The rapid brief auricular contraction now gives place to relaxation, and, just as this begins, the ventricle is seen to become smaller and paler, and, if held in the finger, is felt to become firmer. This event takes place more slowly than the contraction of either sinus or auricles. The chief change in the ventricle is a diminution in its lateral diameter, though it is also decreased in the antero-posterior and vertical directions. During ventricular contraction the bulbus is seen to be distended and to become of a darker colour. The ventricular contraction passes off suddenly, the ventricle again becoming larger and of a deep red colour. At this moment the bulbus aortæ contracts and becomes pale and then relaxes before the next ventricular contraction.

Each chamber of the heart thus passes through two phases—a contraction phase, a *systole* of short duration, and a longer relaxation phase, the *diastole.* And the sequence of events in the frog's heart might be schematically represented as in Fig. 120.

B. *Mammal.*—1. *Rate of Recurrence.*—The rate of recurrence of the cardiac cycle varies with the animal examined. In man it is in adult life about 72 per minute. Many factors modify the rate of the heart.

(1) *Period of Life.*—The following table shows the average rate of the heart at different ages:—

Fœtus	140	per minute.
Under 1 year	120	,,
1 to 3 years	100	,,
7 to 14 years	85	,,
21 to 61 years	70 to 75	,,
Old age	75 to 80	,,

(2) *Period of the Day.*—The pulse is generally slowest in the early morning, and quickest in the evening.

(3) *Temperature of the Body.*—The pulse varies with the body temperature, generally

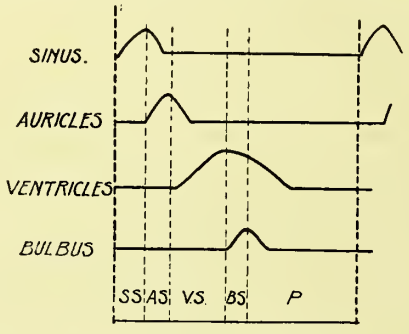


FIG. 120.—Scheme of the cardiac cycle in the frog. *S.S.*, Sinus systole; *A.S.*, auricular systole; *V.S.*, ventricular systole; *B.S.*, bulbus systole; *P.*, rest of all chambers.

being increased about 10 beats with each degree Fahr. of elevation of temperature.

(4) *Muscular exercise* increases the rate of the heart—first, by driving the blood from the muscles into the great veins (p. 37); and second, by developing substances such as CO_2, which act directly upon the cardiac and respiratory mechanisms.

(5) *Posture* has also an important influence. Suddenly assuming the erect position accelerates the heart by causing the blood to accumulate in the abdominal veins, and thus checking its transference on into the arteries (p. 38).

(6) The condition of the central nervous system may modify the rate of the heart, any disturbance accompanied by emotional changes either accelerating or retarding it.

(7) Stimulation of certain nerves—especially those of the abdomen—tends to cause a retardation in the rate of the heart (p. 22).

2. *Sequence of Events.*—The sequence of events making up the cardiac cycle is simpler in the mammal than in the frog.

The contraction starts in the great veins which enter the auricles, and spreads down along them to these chambers. This corresponds to the contraction of the sinus in the frog's heart. It is followed by a short sharp contraction of the auricles, which become smaller in all directions and seem to be pulled down towards the ventricles. The contraction of the auricles in mammals is not accompanied by so

marked a dilatation of the ventricles as in the frog.

After the auricles have fully contracted, the contraction of the ventricles begins, and immediatcly the auricles relax and resume their original size.

The ventricular contraction develops suddenly, lasts for some time, and then suddenly passes off.

The contraction of the ventricles is followed by a period during which both auricles and ventricles remain relaxed. This is called the pause of the cardiac cycle.

The cardiac cycle in mammals may be represented as in Fig. 121.

3. *Duration of the Phases.*—Ventricular systole lasts three times as long as auricular systole;

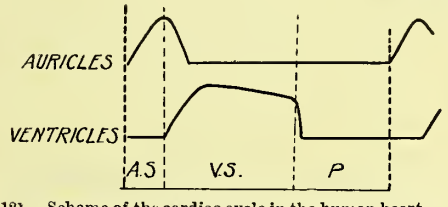


FIG. 121.—Scheme of the cardiac cycle in the human heart. *A.S.*, Auricular systole; *V.S.*, ventricular systole; *P.*, pause.

the auricles contract for about 0·1 of a second, the ventricles for 0·3 of a second.

The duration of these two phases in relationship to the pause varies very greatly. Whatever may be the rate of the heart, the auricular and ventricular systoles do not vary; but in a rapidly acting heart the pause is short, in a slowly acting heart it is long. Taking the ordinary heart rate of 72 per minute, the auricular systole lasts for one-eighth of the whole cardiac cycle, the ventricular for three-eighths, and the pause for four-eighths.

4. *Changes in the Shape of the Chambers.*—(1) *Auricles.*—These simply become smaller in all directions during systole.

(2) *Ventricles.*—The changes in the diameters of the ventricles may be studied by fixing them in the various phases of contraction and measuring the alterations in the various diameters.

The shape in *diastole* may be investigated after death stiffening has passed off and has left the walls relaxed. The condition at the *end of systole* may be studied by rapidly excising *the heart* while it is still beating and plunging it in some hot solution to fix its contraction.

The condition *in the early stage of systole*, before the blood has left the ventricles, may be studied by applying a ligature round the great vessels and then plunging the heart in a hot solution to cause it to contract round the contained blood which cannot escape.

Measurements of hearts so fixed show that at the beginning of contraction the antero-posterior diameter is increased, while the lateral diameter is diminished. In contracting, the lateral walls appear to be pulled towards the septum—the increase in the antero-posterior diameter being largely due to the blood in the right ventricle pressing on and pushing forward the thin wall of the conus.

As the ventricles drive out their blood, both antero-posterior and lateral diameters are diminished—but the diminution in the lateral direction is the more marked.

There is no great shortening in the long axis of the heart. Although the contraction of the longitudinal fibres tends to approximate base and apex, this is in part prevented by the contraction of the circular fibres.

5. *Changes in the Position of the Heart.*—During contraction the heart undergoes, or attempts to undergo, a change in position. In the relaxed condition it hangs downwards and to the left from its plane of attachment, but when it becomes rigid in ventricular contraction it tends to take a position at right angles to its base—*Cor sese erigere*, as Harvey describes the movement. Since the apex and front wall are in contact with the chest, the result of this movement is to press the heart more forcibly against the chest wall. This gives rise to the cardiac impulse which is felt with each ventricular systole over the præcordium (Fig. 119).

If the chest is opened and the animal placed on its back this elevation of the apex is readily seen. If the animal is placed on its belly, so that the heart when relaxed hangs forwards, the apex is tilted back during contraction.

Since the apex is twisted to the left, the movement of the ventricle is not simply directly forward, but also from left to right. This tilting of the apex from left to right is further favoured by the direction of the muscular fibres of the ventricles which pass from the auriculo-ventricular rings downwards and to the left.

The increased thickness of the heart from before backwards also assists, to some extent, in the production of the impulse.

The study of the position and characters of the cardiac impulse is of great importance in medicine.

The *position* is determined by the relationship of the heart to the anterior chest wall and to the lungs. The boundaries of the part of the heart lying in relationship to the chest wall have been already defined (p. 13), and it is at the outer and lower part of this area, a region bounded above by the fifth rib, below by the sixth rib, outside by a line drawn vertically through the nipple, and inside by a line drawn vertically midway between the nipple and the left edge of the sternum, that the cardiac impulse is felt. Normally it does not extend outwards beyond the nipple line, but frequently when the left lung is voluminous the impulse only extends out to an inch or so inside of the nipple line. In children, on account of the size of the liver, it is often felt between the fourth and fifth ribs. This impulse is often called the apex beat—but

it is not the apex which presses on the chest wall but a part of the front of the right ventricle.

In *character* it is felt as a forward impulse of the tissue occupying the fifth interspace, which develops suddenly, persists for a short period, and then suddenly disappears. In many forms of heart disease its character is markedly altered.

The cardiac impulse may be recorded graphically by means of any of the various forms of cardiograph, one of the simplest consisting of a receiving and recording tambour connected by means of a tube (Fig. 122).

The form of the trace varies according to the part of the heart upon which the button is placed, but it has the character shown in Fig. 123 if the button is upon the cardiac impulse.

At the moment of ventricular systole the lever is suddenly thrown up to a certain level

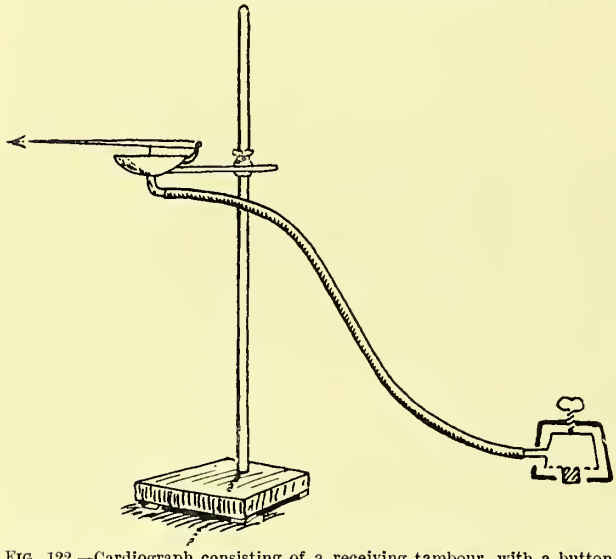


FIG. 122.—Cardiograph consisting of a receiving tambour, with a button on the membrane which is placed upon the cardiac impulse, and a recording tambour connected with a lever.

(*a* to *b*). From this point it suddenly falls slightly (*b* to *c*), but is maintained during the ventricular systole above the abscissa (*c* to *d*). At the end of the ventricular systole, as the heart falls away from the chest wall, the lever falls to its original level (*d* to *e*). In many tracings a small rise of the lever may be seen just before the great upstroke. This corresponds to the contraction of the auricles.

In various diseases of the heart the cardiogram is materially modified. Hence it is important to have a clear conception of the various parts of the trace.

The elucidation of the various parts of the cardiogram is only possible after careful study of the other changes in the heart during the cycle.

6. *Changes in the Intracardiac Pressure.*—These can be studied only in the lower animals.

The most common way of determining the pressure in a cavity is to connect it to a vertical tube and to see to what height the fluid in the cavity is raised. If such a method be applied to the ventricles of the heart, the blood in the tube undergoes such sudden and

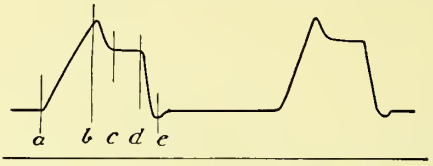


FIG. 123.—Cardiographic trace. *a* to *d*, Ventricular contraction.

enormous changes in level that it is impossible to get accurate results.

The same objection applies to the method of connecting the heart with a U tube filled with mercury. When this is done the changes in pressure are so sudden and so extensive that the mercury cannot respond to them on account of its inertia.

Various means of obviating these difficulties have been devised. One of the best is to allow the changes of pressure to act upon a small elastic membrane tested against known pressures. A tube is thrust through the wall of the heart and connected with a tambour covered by a membrane to which a lever is attached.

A. Pressure in the Great Veins (small dotted line in Fig. 124).—When the auricles contract, the flow of blood from the great veins into these chambers is arrested, and, as a result, the pressure in the veins rises. As the auricles relax the blood is sucked from the veins and the pressure falls, but as the auricles fill up it again rises. When the ventricles relax and suck blood from the auricles, blood again flows in from the great veins and the pressure falls, again to rise as the auricles and veins are both filled up, towards the end of the pause.

B. Pressure in the Auricles (dash line in Fig. 124).—At the moment of auricular contraction there is a marked rise in the intra-auricular pressure. When the auricular systole stops, the pressure falls rapidly, reaching its lowest level when the ventricles are throwing their blood into the arteries. From this point the pressure in the auricles rises until the moment when the ventricles relax, when another fall in the pressure is observed. The pressure remains about constant from this point until the next auricular contraction.

C. Pressure in the Ventricles (continuous line in Fig. 124).—The intra-ventricular pressure suddenly rises at the moment of ventricular systole to reach its maximum. From this it falls, but the fall is gradual, and is interrupted by a more or less well-marked period during

which the pressure remains constant. As the ventricles relax the pressure suddenly falls to below zero, and then rises to a little above zero, at which it is maintained until the next ventricular systole. The diastolic expansion of the ventricle is in part due to the elasticity of the muscular wall, and in part to the filling of the coronary arteries which takes place only as the muscular fibres relax.

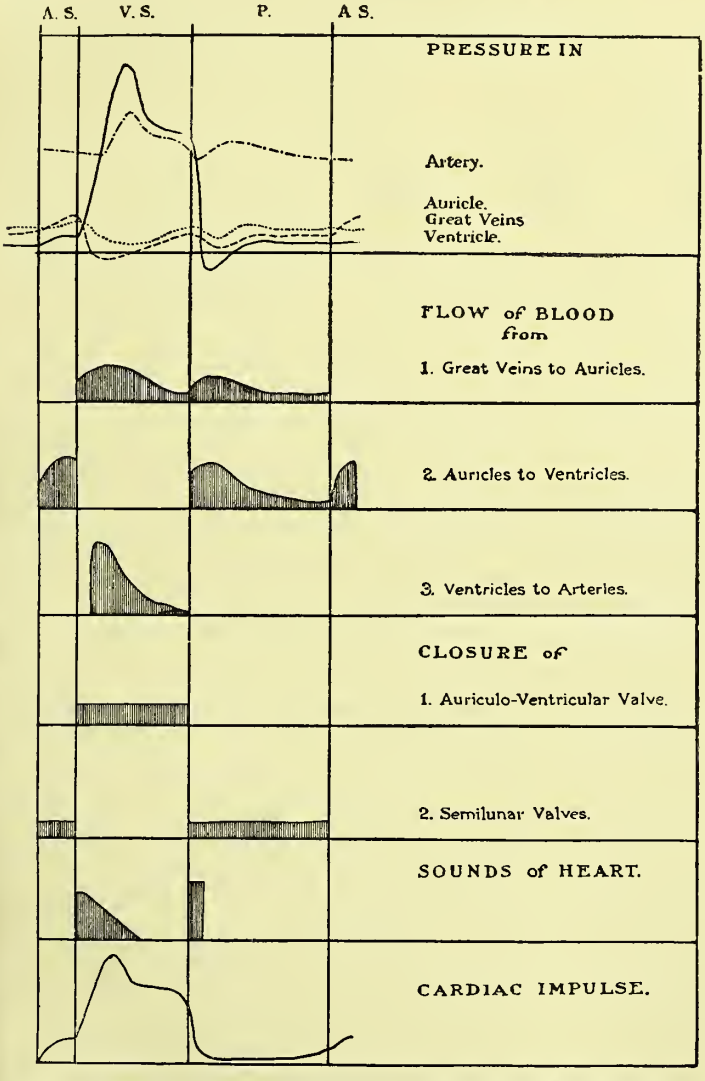


FIG. 124.—Diagram to show the relationship of the events in the cardiac cycle to one another. *A.S.*, Auricular systole; *V.S.*, ventricular systole; *P.*, pause.

D. Pressure in the Arteries (dot-dash line in Fig. 124).—The aortic pressure is high throughout. There is a sudden rise as the blood rushes out of the ventricles. The pressure then falls, but the fall is not steady. Often it is interrupted by a more or less marked increase corresponding to the later part of the ventricular contraction. At the moment of ventricular diastole, the fall is very sharp and is interrupted by a well-marked and sharp rise. Following this the fall is continuous till the next systolic elevation.

In the dog the extent of variation of the pressure in auricles and ventricles is roughly as follows—measured in millimetres of mercury—

	Left Ventricle.	Right Ventricle.	Right Auricle.
Maximum . .	+140	+60	+30
Minimum . .	− 30	− 15	− 7

These changes in the pressure in the different chambers are due—

1*st.* To the alternate systole and diastole of the chambers, the first raising, the second lowering the pressure in the chambers.

2*nd.* To the action of the valves.

7. *Action of the Valves of the Heart.—A. Auriculo-ventricular* (Fig. 125).—These valves have been already described as funnel-like prolongations of the auricles into the ventricles. They are firmly held down in the ventricular cavity by the chordæ tendineæ. When the ventricle contracts the papillary muscles pull the cusps of the valves together and thus occlude the opening between auricles and ventricles. The cusps are further pressed face to face by the increasing pressure in the ventricles, and they may become convex towards the auricles. They thus form a central core around and upon which the ventricles contract.

On the *left side* of the heart the strong anterior cusp of the mitral valve does not materially shift its position. It may be somewhat pulled backwards and to the left. The posterior cusp is pulled forwards against the anterior.

On the *right side* the infundibular cusp of the tricuspid valve is stretched between the superior and inferior papillary muscles, and is thus pulled towards the bulging septum, against which it is pressed by the increasing pressure inside the ventricles. The posterior cusp has its anterior margin pulled forward and its posterior margin backwards, and is thus also pulled toward the septum. The septal cusp remains against the septum. The greater the pressure in the ventricle, the more firmly are these cusps pressed against one another and against the septum, and the more completely is the orifice between the auricle and the ventricle closed. On the right side of the heart other factors play an important part in occlud-

ing the orifice; the muscular fibres which surround the auriculo-ventricular opening contract, while the papillary muscles pull the auriculo-ventricular ring downwards and inwards through the chordæ which are inserted into it.

Nevertheless the occlusion of this orifice is apt to be incomplete when the right side of the heart becomes in the least over-distended, giving rise to a safety-valve action from the right ventricle.

The auriculo-ventricular valves are open during the whole of the cardiac cycle, except during the ventricular systole (Fig. 124).



FIG. 125.—State of the various parts of the heart throughout the cardiac cycle. 1, Auricular systole; 2, beginning of ventricular systole (latent period); 3, period of outflow from the ventricle; 4, period of residual contraction; 5, beginning of ventricular diastole.

B. Semilunar Valves.—Before the ventricles contract these valves are closed and the various segments pressed together by the high pressure of blood in the aorta.

As the ventricles contract the pressure in them rises, until the intra-ventricular pressure becomes greater than the pressure in the arteries. Instantly the cusps of the valves are thrown back, and remain thus until the blood is expelled. When the outflow of blood is completed, the cusps are again approximated by the pressure of blood in the arteries. As relaxation of the ventricles occurs, the intra-ventricular pressure becomes suddenly very low, and the high pressure of the blood in the arteries at once falls upon the upper surfaces of the cusps, which are thus forced downwards and together, and thus completely prevent any back-flow of blood.

The prejudicial effect of too great pressure upon these cusps is obviated by the lower cusp being mounted on the top of the muscular septum upon which the pressure falls—the other cusps shutting down upon this one (Fig. 118).

THE FLOW OF BLOOD THROUGH THE HEART.—The circulation of blood through the heart depends upon these differences of pressure in the different chambers and upon the action of the valves.

A fluid always flows from a point of high pressure to a point of lower pressure. We may then consider the flow—

A. From Great Veins into Auricles. — This occurs when the pressure in the great veins is greater than the pressure in the auricles (Fig. 124).

The pressure in the auricles is lowest at the moment of their diastole. At this time there is therefore a great flow of blood into them, but gradually this becomes less and less, until, when the ventricles dilate, another fall in the auricular pressure takes place and another rush of blood from the great veins occurs. Gradually this diminishes, and by the time that the auricles contract the flow from the great veins has stopped.

The contraction of the mouths of the great veins, which precedes the auricular systole, drives blood from the veins into the auricles, and, as these enter into contraction, no flow from the veins can occur and no back-flow from the auricles is possible (Fig. 124).

B. From Auricles to Ventricles. — As the ventricles dilate, a very low pressure develops in them, and hence a great rush of blood occurs from the auricles. During the passive stage of ventricular diastole, the intra-ventricular pressure becomes nearly the same as the auricular, and the flow diminishes or may stop. When the auricles contract a higher pressure is developed, and a fresh flow of blood occurs into the ventricles. When the ventricles contract the auriculo-ventricular valves are closed, and all flow of blood from the auricles is stopped (Fig. 124).

C. From Ventricles to Arteries.—When the ventricles begin to contract the intra-ventricular pressure is low, while the pressure in the arteries is high and keeps the semilunar valves shut. As ventricular systole goes on the intra-ventricular pressure rises, until after about 0·03 of a second it becomes higher than the arterial pressure (*Latent Period*). Immediately the semilunar valves are forced open and a rush of blood occurs from the ventricles (*Period of Overflow*). This usually lasts less than 0·2 second. If the ventricles are acting powerfully, and if the pressure in the arteries does not offer a great resistance to the entrance of blood, *the ventricles rapidly empty themselves* into the arteries, and the intra-ventricular pressure varies as shown in Fig. 132, *b*, p. 27. If the heart, however, is not acting forcibly, or if the arterial pressure offers a great resistance to the entrance of blood, then *the outflow is slow and more continued*, and in this case the trace of the intra-ventricular pressure is like Fig. 132, *a*, with a well-marked *Period of Residual Contraction.* It is not so much the *absolute* force of the cardiac contraction or the *absolute* intra-arterial pressure which governs this, as the relationship of the one to the other. The heart may not be acting very forcibly, but still if the pressure in the arteries is low its action may be *relatively* strong.

The Coronary Arteries, unlike all the other arteries, are filled during ventricular diastole. During systole they are compressed by the

contracting muscle of the heart, and it is only when the compression is removed in diastole that blood rushes into them. This helps to dilate the ventricles.

The interpretation of the various details of the cardiogram is now rendered more easy. The ventricles, still full of blood, are suddenly pressed against the chest wall in systole. As the blood escapes into the arteries they press with less force, and hence the sudden slight downstroke (Fig. 123, *b* to *c*). But, so long as the ventricles are contracted, the apex is kept tilted forward, and hence the horizontal plateau is maintained (*c* to *d*). The pressure of the apex disappears as the ventricles relax (*e*).

Sounds of the Heart.—On listening in the region of the heart, a pair of sounds may be heard with each cardiac cycle, followed by a somewhat prolonged silence. These are known respectively as the First and Second Sounds of the Heart (Fig. 124).

By placing a finger on the cardiac impulse while listening to these sounds it is easy to determine that the first sound occurs synchronously with the cardiac impulse—*i.e.* synchronously with the ventricular contraction.

It develops suddenly, and dies away more slowly. In character it is dull and rumbling, and may be imitated by pronouncing the syllable lūb. In pitch it is lower than the second sound.

The second sound is heard at the moment of ventricular diastole. Its exact time in the cardiac cycle has been determined by recording it on the cardiac tracing by means of a microphone. It develops suddenly and dies away suddenly. It is a clearer, sharper, and higher-pitched sound than the first. It may be imitated by pronouncing the syllable dŭpp.

According to the part of the chest upon which the ear is placed, these sounds vary in intensity. Over the apical region the first sound is louder and more accentuated; over the base the second sound is more distinctly heard.

The *Cause of the Second Sound* is simple. At the moment of ventricular diastole, when this sound develops, the only occurrence which is capable of producing a sound is the sudden stretching of the semilunar valves by the high arterial pressure above them and the low intraventricular pressure below them. The high arterial pressure comes on them suddenly like the blow of a drum-stick on a drum-head, and, by setting the valves in vibration, produces the sound.

Aortic and Pulmonary Areas.—The second sound has thus a dual origin—from the aortic valve and from the pulmonary valve; and it is possible by listening in suitable positions to distinguish the character of each of these.

The aortic valve is placed behind the sternum at the level of the lower border of the third costal cartilage. But it is deeply situated. The aorta, passing upwards and forwards, lies in close relationship to the chest wall at the junction of the right side of the sternum and the right second costal cartilage. The sound produced by the valve is conducted up the aorta, and may be heard best in this "aortic area."

On the other hand, the pulmonary valve lies in close relationship to the anterior chest wall—being covered only by the anterior border of the left lung—close to the edge of the sternum in the second left interspace. The pulmonary element of the second sound may best be heard here.

The *Cause of the First Sound* is by no means so simple. When it is heard, two changes are taking place in the heart, either of which would produce a sound.

1*st.* The muscular wall of the ventricles is contracting.

2*nd.* The stretching of the auriculo-ventricular valves.

1*st.* That the first factor plays an important part in the production of the first sound is proved by rapidly cutting out the heart of an animal, and while it is still beating—but without any blood passing through it to stretch the valves—listening to the organ with a stethoscope. With each beat the lūb sound is distinctly heard.

Apparently the wave of contraction, passing along the muscular fibres of the heart, sets up vibrations, and when these are conducted to the ear the external meatus picks out the vibration corresponding to its fundamental note, and thus produces the characters of the sound.

2*nd.* The auriculo-ventricular valves are being closed and subjected on the one side to the high ventricular pressure and on the other to the low auricular pressure. If the valves be destroyed or diseased the characters of the first sound are materially altered, or the sound may be entirely masked by a continuous musical sound—a murmur. Again, it has been maintained that a trained ear can pick out in the first sound the note corresponding to the valvular vibration.

The idea that the impulse of the heart against the chest wall plays a part in the production of this sound is based upon the fallacious idea that the heart "*hits*" the chest wall. All that it does is to press more firmly against it.

Mitral and Tricuspid Areas.—On account of the part played by the valves in the production of the first sound, it may be considered to be double in nature—partly due to the mitral valve, partly to the tricuspid. The mitral valve element may best be heard *not* over the area of the mitral valve—which lies very deep in the thorax—but over the apex of the heart, as at this situation the left ventricle, in which the valve lies, comes nearest to the thoracic wall and conducts the sound thither. The

tricuspid element may be best heard over the area of the valve, and in listening to it it is usual to go to the right extremity of the area in order, as far as possible, to eliminate the mitral sound. The best situation to select is at the junction of the fifth right costal cartilage with the sternum.

Cardiac Murmurs. — When the valves are diseased and fail to act properly, certain continuous sounds called cardiac murmurs are heard.

These owe their origin to the fact that, while a current of fluid passing along a tube of fairly uniform calibre is not thrown into vibrations and therefore produces no sound, when any marked alterations in the lumen of the tube occurs—either a sudden narrowing or a sudden expansion—the flow of fluid becomes vibratory, and, setting up vibrations in the solid tissues, produces a musical sound.

Such changes in the calibre of the heart are produced in two ways:—

1*st.* By a narrowing, either absolute or relative, of the orifices between the cavities—*stenosis.*

2*nd.* By a non-closure of the valves—*incompetence.*

Stenosis.—If one of the auriculo-ventricular orifices is narrowed, a murmur is heard during the period at which blood normally flows through this opening. A reference to Fig. 124 at once shows that this occurs during the whole of ventricular diastole, and that the flow is most powerful during the first period of ventricular diastole and during auricular systole.

If the aortic or the pulmonary valve is narrowed the murmur will be heard (Fig. 124) during ventricular systole.

The narrowing need not be absolute. A dilatation of the artery will make the orifice *relatively* narrow, and will produce the same result.

Incompetence. — If the auriculo-ventricular valves fail to close properly, then, during ventricular systole, blood will be driven back into the auricles, and a murmur will be heard during this period.

If the aortic or pulmonary valve fails to close, the blood will regurgitate into the ventricle from the arteries during ventricular diastole, and a murmur will be heard during this period.

By the position at which these murmurs are best heard the pathological condition producing them may be determined.

Work of the Heart.—The heart in pumping blood through it is doing work, and the amount of work may be expressed in work units—*e.g.* kilogrammetres (*see* vol. vii. p. 511). With each beat something under 80 grms. of blood are thrown from each ventricle into the aorta and pulmonary artery. Thus the weight lifted is about 0·08 kilos. The output of blood at each beat of the heart of the dog may be measured by Roy's cardiometer, a rigid-walled air-tight case, which is placed round the heart and connected with a piston-recorder, so that the decrease in the volume of the enclosed heart due to the blood leaving it may be directly recorded by means of a lever attached to the piston (Fig. 126).

The average resistance in the aorta may be taken at about 1·5 metres of blood. Hence, with each beat, the left ventricle may perform $0{\cdot}08 \times 1{\cdot}5 = 0{\cdot}12$ kgms. of work. The right

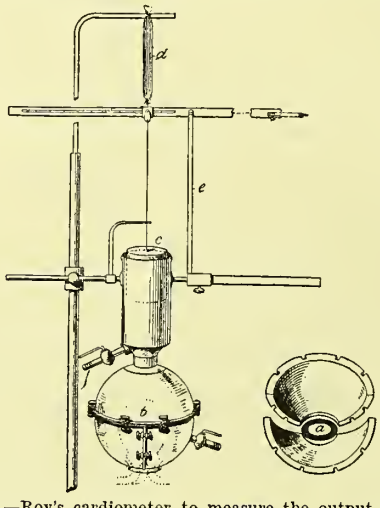


Fig. 126.—Roy's cardiometer to measure the output of blood from the heart. *b*, Heart in cardiometer chamber; *e*, piston recorder working on lever against rubber band, *d*.

ventricle is only one-third as strong as the left, and hence the work done by each beat is only 0·04 kgms.

If the heart is beating seventy-two times per minute, the amount of work will be something under 11·5 kgms. per minute, or 16,560 kgms. in twenty-four hours. Some investigators estimate it as low as 10,000 kgms.

Nature of Cardiac Contraction.—The contraction of the ventricle lasts for a considerable period—0·3 seconds. Is it of the nature of a single contraction, or of a tetanus?

It is impossible to tetanise heart muscle, even by rapidly repeated induction shocks. A single stimulus applied to heart muscle produces a single prolonged contraction. Again, the mode of development of the currents of action does not indicate anything of the nature of a tetanus. With each beat of the ventricles the variation in the electric potential begins at the base and travels rapidly to the apex. This passage of the contraction wave along the fibres explains the great length of the ventricular systole as a whole. There can be no doubt that each contraction of heart muscle is of the nature of a muscle twitch. In this respect heart muscle resembles non-striped muscle (*see* vol. vii. p. 515).

It further resembles it in that the minimum stimulus is also a maximum stimulus—*i.e.* the smallest stimulus which will make the muscle contract makes it contract to the utmost. But

while this is the case the strength of stimulus necessary to call forth a contraction varies at different periods. To produce another contraction while the muscle is already in the period of contraction is difficult, but as it relaxes it reacts more and more readily to stimuli. In cardiac muscle, perhaps more than in any other, the staircase increase in the extent of contraction with a series of stimuli is manifested.

In cardiac muscle the *greater the resistance to contraction the stronger the force of contraction.* Hence when extra blood is poured into the heart from the veins, or when the outflow from the ventricles into the arteries is impeded, the increased strain put upon the heart muscle is met by increased contraction, and the additional work thrown upon the organ is effectually performed. Not only is this the case when temporary disturbances of the circulation occur, but when these disturbances are permanent, the heart adapts itself to them, and, if it has continuously to perform extra work, its muscular wall hypertrophies, just as the skeletal muscles grow by continual use. Of course, to allow such compensation to be established, the blood supply to the heart muscle must be sufficient, and hence, when the coronary arteries are diseased, heart failure rapidly ensues.

If the coronary arteries are clamped and then relaxed, a peculiar fibrillar contraction of the heart muscle occurs.

The maintenance of this rhythmic contraction and relaxation seems to depend greatly upon the presence of certain kations in the circulating blood. A due admixture of salts of sodium, potassium, and calcium is essential. For the frog's heart Ringer finds that the proportions which give the best results are—

NaCl	. . .	0·70 per cent.
KCl	. . .	0·03 ,,
CaCl	. . .	0·025 ,,

Since an excess of calcium salts leads to tonic contraction, and since an excess of sodium or of potassium leads to relaxation, it has been concluded that these two phases are determined by the presence of these ions.

How is the Rhythmic Contraction of the Heart maintained?—The mechanism is *in the heart itself*, for the excised heart continues to beat.

In considering what this mechanism is, it must be borne in mind that two distinct questions have to be investigated.

1*st.* How does the contraction, once started, pass in regular sequence from one part of the heart to the other?

2*nd.* What starts each rhythmic contraction?

1*st. Propagation of the Wave of Contraction.*—In the heart of many of the lower animals, and in the embryo of mammals, no nervous structures are to be found, and the rhythmic contraction is manifestly simply a function of the muscular fibres.

Even in the heart of animals with well-marked nerve cells in the walls of the heart, and with nerve fibres coursing among the muscular fibres, the conduction of the contraction is purely a function of the muscles. For if the heart of a frog be cut across and across, so that all nerve fibres are severed, the contraction passes along it. The rate at which the contraction travels is slow, only about 10 to 15 centimetres per second.

Since in the mammalian heart muscular continuity between auricles and ventricles through the band of His is of small extent, the wave of contraction is delayed at this point, and in the dying heart, and in various pathological conditions, the contraction frequently fails altogether to pass this block, and thus the ventricles either stop beating before the auricles, or respond to every second or third auricular contraction.

2*nd. Starting Mechanism of Contraction.*—In the early fœtal heart no nerve structures have been found, yet it beats regularly and rhythmically. In the apex of the ventricle of the frog there are no nerve structures, yet, if the apex be cut off and repeatedly stimulated at regular intervals with galvanic making and breaking stimuli, it will, after a time, begin to contract spontaneously, regularly and rhythmically. Not only so, but if the apex be tied on to a tube, and a stream of blood passed through it, it will again start contracting regularly and rhythmically.

These experiments clearly show that regular rhythmic contraction is a function of cardiac muscle.

In the cardiac cycle in the frog each contraction starts in the sinus. What part does the sinus take in initiating contraction?

If a ligature be tightly applied between the sinus and auricles in the frog (*Stannius' Experiment*), the sinus continues to beat, and the auricles and ventricle *usually* stop beating for a longer or shorter period. But ultimately they begin to beat again. Hence it would seem that it is not any special mechanism in the sinus which is essential in starting cardiac contraction. A ligature subsequently applied between auricles and ventricle sometimes starts the ventricle, sometimes the auricles, sometimes neither. Hence we see *that any part of the heart has the power of originating rhythmical contractions*, although usually the sinus initiates it. The sinus more than any other part of the heart has the property of rhythmic contraction.

We have no evidence that the nerve cells in the sinus or elsewhere have anything to do with this; and, so far as we at present know, the initiation as well as the propagation of the cardiac contraction is a function of the muscular fibres.

3*rd. Intra-cardiac Nervous Mechanism.*—In the frog's heart nervous structures exist, and are distributed as follows (Fig. 127):—

1*st.* In the wall of the sinus venosus there

is a plexus of nerve cells and nerve fibres constituting the ganglion of the sinus (Remak's ganglion).

2*nd*. In the inter-auricular septum a similar plexus constitutes the ganglion of the auricular septum.

3*rd*. In the auriculo-ventricular groove a plexus forms the auriculo-ventricular ganglion (Bidder's ganglion). With these intra-cardiac ganglia the terminations of the nerves to the heart form definite synapses.

Nerve cells exist in the mammalian heart, but there is not the same differentiation into distinct groups. Nevertheless they are abundant round the mouths of the great veins, round the edges of the inter-auricular septum, and round the auriculo-ventricular groove.

While there is no evidence that the nervous structures play an important part in starting or keeping up the contractions, there is evidence

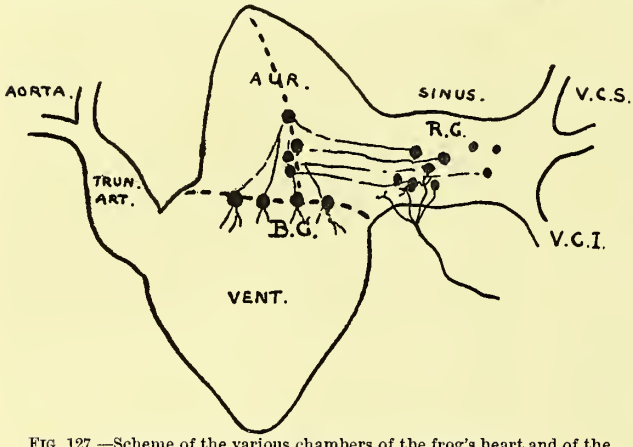


FIG. 127.—Scheme of the various chambers of the frog's heart and of the distribution of the intra-cardiac nervous mechanism.

that they exercise a checking or controlling action.

If the region between the sinus and auricles in the frog's heart is stimulated by the interrupted current from an induction coil, the heart is slowed or stopped.

If atropine be first applied electric stimulation is without result.

These experiments seem to indicate that *there is in the heart a checking mechanism which may be stimulated by electricity, and which is paralysed by atropine.*

4*th*. *Connections of the Heart with the Central Nervous System.*—In the frog a branch from the vagus connects the central nervous system with the heart. When the branch is cut no effect is produced, showing that it is not constantly in action; but when the lower end is stimulated, the heart is generally slowed or brought to a standstill. Sometimes the effect is not marked. The reason for this is that the cardiac branch of the vagus in the frog is really a double nerve derived in part from the spinal accessory and in part from fibres which reach the vagus from the superior thoracic sympathetic ganglion. If the spinal accessory is stimulated, the heart is always slowed; and if the sympathetic fibres are stimulated, it is quickened. Generally stimulation of the cardiac branch containing these two sets of fibres simply gives the result of stimulating the former, but sometimes the stimulation of the latter masks this effect.

In the mammal three sets of nerve fibres pass to the heart:—

1*st*. The superior cardiac branch of the vagus starts from near the origin of the superior laryngeal nerve, and passes to the heart to end in the endocardium (Fig. 128, *S.C.*).

2*nd*. The inferior cardiac branch of the vagus leaves the main nerve near the recurrent laryngeal, and passes to join the superficial cardiac plexus in the heart (Fig. 128, *I.C.*).

3*rd*. The sympathetic nerve fibres come from the superior thoracic and inferior cervical ganglia, and also end in the superficial cardiac plexus (Fig. 128, *S.*).

Functions of the Cardiac Nerves.—A. The *Superior Cardiac Branch of the Vagus* is an ingoing nerve. Section produces no effect; stimulation of the lower end causes no effect; stimulation of the upper end causes slowing of the heart and a marked fall in the pressure of blood in the arteries, and it may cause pain. The slowing of the heart is a reflex effect through the inferior cardiac branch; and the fall of blood pressure, which is the most manifest effect, is due to a reflex dilatation of the vessels of the abdomen, causing the blood to accumulate there, and thus to lessen the pressure in the arteries generally. On account of its effect on the blood pressure, this nerve is called *the depressor nerve.*

B. *Inferior Cardiac Branch of Vagus.*—Section of the vagus or of this branch causes acceleration of the action of the heart. The nerve is therefore constantly in action. Stimulation of its central end has no effect; stimulation of its peripheral end causes a slowing or stoppage of the heart. It is therefore the checking or inhibitory nerve of the heart.

1. *Course of the Fibres.*—These fibres leave the central nervous system by the spinal accessory, and pass to the heart to form connections with the cells of the cardiac plexuses.

2. *Centre.*—The fibres arise from a centre in the medulla oblongata, which can be stimulated to increased activity either directly or reflexly. (1) *Direct stimulation* is brought about by (*a*) sudden anæmia of the brain, as when the arteries to the head are clamped or occluded; (*b*) increased venosity of the blood, as when respiration is interfered with; (*c*) the concurrent action of the respiratory centre (*see* p. 43). (2) *Reflex stimulation* is produced through many nerves. In the rabbit stimulation of the 5th

cranial nerve by the inhalation of ammonia vapours has this action, and in all animals stimulation of the abdominal nerves produces the same effect. This reflex stimulation of the centre is used to determine its position in the medulla. It can be induced after removal

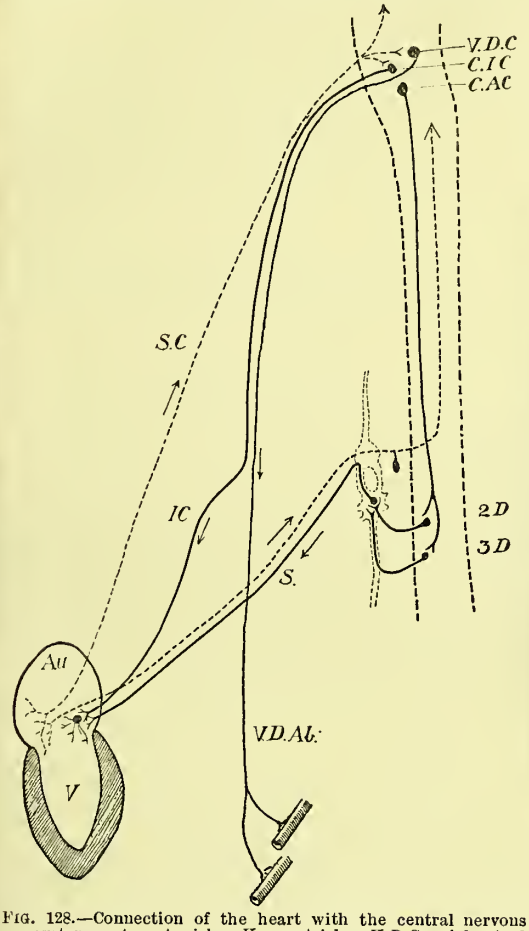


FIG. 128.—Connection of the heart with the central nervous system. *Au.*, Auricle; *V.*, ventricle; *V.D.C.*, abdominal vaso-dilator centre; *C.I.C.*, cardiac inhibitory centre; *C.A.C.*, cardio-augmentor centre; *S.C.*, superior cardiac branch of the vagus; *I.C.*, inferior cardiac branch of the vagus with cell station in the heart; *S.*, cardio-sympathetic fibres with cell station in the lenticular ganglion; *V.D.Ab.*, vaso-dilator fibres to abdominal vessels. The continuous lines are outgoing, the broken lines are ingoing nerves.

of the brain above the medulla, but destruction of the medulla entirely prevents it.

3. *Mode of Action.*—These inhibitory fibres appear to act by stimulating the local inhibitory mechanism in the heart; and when this has been poisoned by atropine, they cannot act. According to the observation of Gaskell, they excite in the heart anabolic changes, since the electric current of injury is increased when they are stimulated, indicating that the difference between the living part of the heart and the injured part is increased (*see* vol. vii. p. 512).

4. *Result of Action.*—(*a*) The output of blood from the heart is diminished, and thus less blood is forced into the arteries, and the blood pressure falls (Fig. 134).

(*b*) The rhythm of both auricles and ventricles is slowed, but the effect on the auricles is more marked than upon the ventricles, and the ventricles may show a contraction rhythm independent of that of the auricles (Fig. 129, *A.*).

(*c*) The force of contraction of the auricles is decreased. In the ventricles the systole becomes less complete and the cavities become more and more distended, either as a result of decrease in the force of contraction or as a mechanical result of the accumulation of blood due to the decreased output per unit of time. In the heart of the tortoise excitability and conductivity are decreased, and the auricular contraction may fail to pass to the ventricles.

C. Sympathetic Fibres.—The *outgoing* fibres are the augmentors and accelerators of the heart's action. When they are cut the heart may beat slower. When the peripheral end is stimulated, the rate and force of the heart are increased.

1. *Course of the Fibres.*—These are small medullated fibres. They leave the spinal cord by the anterior roots of the 2nd, 3rd, and 4th dorsal nerves, passing to the stellate ganglion where they have their cell stations (Fig. 128). From the cells in this ganglion non-medullated fibres run on in the annulus of Vieussens, and from this and from the inferior cervical ganglion they pass out to the muscular fibres of the heart.

2. *The Centre* is in the medulla, and it may be stimulated by stimulating various ingoing nerves, such as the sciatic; or it may be set in action from the higher nerve centres in various emotional conditions.

3. *Mode of Action.*—The fibres seem to act (*a*) upon the muscular fibres, increasing their excitability and conductivity; (*b*) upon the inhibitory mechanism, throwing it out of action.

4. *Result of Action.*—(*a*) The output of blood from the heart is increased, and the pressure of blood in the arteries is raised.

(*b*) The rate of the rhythmic movements of auricles and ventricles is increased.

(*c*) The force of contraction of auricles and ventricles is increased.

It is probable that the cardiac sympathetic also carries *ingoing fibres* which enter the cord in the lower cervical region. The pain experienced in the arm in heart disease is generally thought to be due to the implication of these fibres leading to the sensation which is referred to the corresponding somatic nerves (vol. vii. p. 551).

The vagus is thus the protecting nerve of the heart, reducing its work and diminishing the pressure in the arteries.

The sympathetic is the whip which forces the heart to increased action in order to keep up the pressure in the arteries.

III. *Circulation in the Blood and Lymph Vessels*

The general distribution of the various vessels—arteries, capillaries, veins, and lym-

phatics—has been already considered (Fig. 115, p. 10).

Structure.—(*The structure of the walls of each must be studied practically.*) The capillaries are minute tubes of about 12 micromillimetres in diameter, forming an anastomosing network throughout the tissues. Their wall consists of a single layer of endothelium. On passing from the capillaries to arteries on the one side, and to veins and lymphatics on the other, non-striped muscle fibres make their appearance encircling the tube. Between these fibres and the endothelium a fine elastic membrane next appears, while outside the muscles a sheath of fibrous tissue develops. Thus the three essential coats of these vessels are produced :—

Tunica intima, consisting of endothelium set on the internal elastic membrane.

Tunica media, consisting chiefly of the visceral muscular fibres.

Tunica adventitia, consisting of loose fibrous tissue.

The coats of the arteries are thick; those of the veins are thin. In the large arteries the muscular fibres of the media are largely replaced by elastic fibres, so that the vessels may better stand the strain of the charge of blood which is shot from the heart at each contraction. In the veins, double flaps of the tunica intima form valves which prevent any regurgitation of blood.

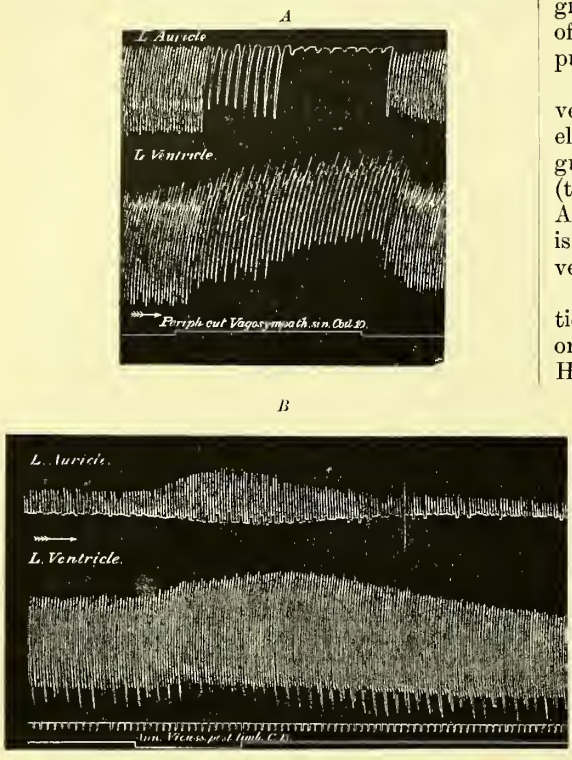


Fig. 129.—Simultaneous tracing from auricles and ventricles. *A.*, During stimulation of the vagus; *B.*, during stimulation of the sympathetic. Each downstroke marks a systole, each upstroke a diastole. (From Roy and Adam.)

The great characteristic of the walls of the *large arteries* is the toughness and elasticity given by the abundance of elastic fibrous tissue; of the *small arteries*, the contractility due to the preponderance of muscular fibres.

Physiology.—The circulation of blood in the vessels is that of a fluid in a closed system of elastic-walled tubes, at one end of which (the great arteries) a high pressure, and at the other (the great veins) a low pressure, is kept up. As a result of this distribution of pressure there is a constant flow of blood from arteries to veins.

Many points in connection with the circulation may be conveniently studied on a model or scheme made of indiarubber tubes and a Higginson's syringe.

A.—Blood Pressure

The distribution of pressure is the cause of the flow of blood, and must first be considered.

I. General Distribution of Pressure.—That the pressure throughout the greater part of the blood-vessels is positive—greater than the pressure of the atmosphere—is indicated by the fact that if a vessel is opened, the blood flows out of it. *The force with which blood escapes is a measure of the pressure in that particular vessel.* If an artery be cut, the blood escapes with great force; if a vein be cut, with much less force (Fig. 130).

Arteries.—If the pressure in the aorta, in the radial, in the dorsalis pedis, and in one of the smallest arteries is measured, it is found that while it is great in the great arteries—about 160 mm. Hg in the aorta—it is much less in the small arteries. This distribution of arterial pressure might be plotted out as in Fig. 130, *Ar.*

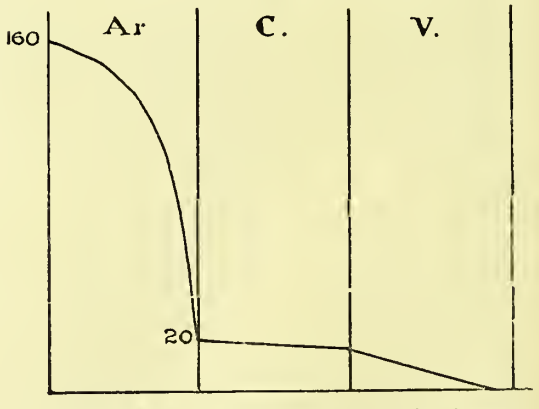


Fig. 130.—Diagram of the distribution of mean blood pressure throughout the blood-vessels. *Ar.*, The arteries; *C.*, the capillaries; *V.*, the veins.

Veins.—If the pressure in any of the small

veins, in a medium vein, and in a large vein near the heart, be measured, it will be found—

1*st.* That the venous pressure is less than the lowest arterial pressure.

2*nd.* That it is highest in the small veins, and becomes lower in the larger veins. In the great veins entering the heart it is lower than the atmospheric pressure during the first part of each ventricular diastole (Fig. 130, *V.*).

Capillaries.—The pressure in the capillaries must obviously be intermediate between that in the arteries and in the veins.

It is not so easily measured, but it may be approximately arrived at by finding the pressure which is required to empty the capillaries—*e.g.* to blanch a piece of skin.

The pressure in any part of a system of tubes depends upon two factors—

1*st.* The force propelling fluid into that part of the tubes.

2*nd.* The resistance to the outflow of fluid from that part of the tubes.

The pressure in the arteries is high, because with each beat of the heart about 80 grms. of blood are thrown with the whole contractile force of each ventricle into the corresponding artery; and because the resistance offered to the outflow of blood from the arteries into the capillaries and veins is enormous. For, as the blood passes into innumerable small vessels, it is subjected to greater and greater friction—just as a river in flowing from a deep narrow channel on to a broad shallow bed is subjected to greater friction.

Thus in the arteries the powerful propulsive force of the heart and the great resistance to outflow keep the pressure high.

When the capillaries are reached much of the force of the heart has been lost in dilating the elastic coats of the arteries, and thus the inflow into the capillaries is much weaker than the inflow into the arteries. At the same time the resistance to outflow is small, for in passing from capillaries to veins the channel of the blood is becoming less broken up, and thus opposes less friction to the inflow of the blood.

When the veins are reached the propelling force of the heart is still further weakened, and hence the force of inflow is very small. But, instead of there being a *resistance* to outflow from the veins into the heart, this is favoured by the suction action of the heart during diastole, and also by the fact that the great veins, in entering the heart, pass into the thorax, an air-tight box in which during each inspiration a very low pressure is developed.

What has been said of the veins applies equally to the lymphatics.

2. Rhythmic Variations in Blood Pressure.—Before considering the exact measurements of pressure in these different vessels, certain rhythmic variations in pressure may first be considered.

A. *Synchronous with the Heart Beats*

Arterial Pulse.—With each ventricular systole about 80 grms. of blood are thrown into the already full arteries, and the pressure in these vessels is suddenly raised.

If the finger be placed on an artery, a distinct expansion will be felt following each systole, and due to this rise of pressure. This is the arterial pulse.

This expansion develops suddenly and disappears more slowly. In some cases it may be felt by simply laying the finger on the surface of the artery without exerting marked pressure, in other cases it may be necessary to compress the artery before the pulsation is distinctly felt.

If a vein be investigated in the same way it will be found that no such pulse can be detected. In the capillaries, too, this pulse does not exist.

It is best marked in the great arteries, and becomes less and less distinct as the small terminal arteries are reached.

Cause of Pulse.—The arterial pulse is due to—

1*st.* The intermittent inflow of blood. The arteries expand from the sudden increase of pressure due to each sudden flow of 80 grms. of blood from the heart into the arterial system.

2*nd.* The resistance to outflow from the arteries into the capillaries.

If blood could flow freely from the arteries into the capillaries, then the inrush of blood from the heart would simply displace the same amount of blood into the capillaries and the arteries would not be expanded. As already indicated, the friction between the walls of the innumerable small arterioles and the blood is so great that the flow out of the arteries is not so free as to allow the blood to pass into the capillaries so rapidly as it is shot into the arteries. Hence, with each beat of the heart, an excess of blood must accumulate in the arteries.

3*rd.* To allow of their expanding to accommodate this excess of blood their walls must be *elastic.*

It is upon these three factors that the arterial pulse depends. Do away with any of them, and the pulse at once disappears.

Why is there no Pulse in the Veins?—Their walls are elastic, but, in the first place, instead of there being an obstruction to the outflow of blood from the veins into the heart, this is favoured by the suction action of the heart and thorax. Hence, even if an intermittent inflow were well marked, the absence of resistance to outflow would in itself prevent the development of a venous pulse. But the inflow is not intermittent. With each beat of the heart the blood does not pass freely from the arteries into the capillaries and veins, but it only slowly escapes, *just as much passing out between the beats as during the beats.* Hence the most important factor in causing a pulse, an intermittent inflow, is absent.

With no sudden intermittent inflow, and with no resistance to outflow, the development of a pulse is impossible.

In certain abnormal conditions, where, from the extreme dilatation of the arterioles, the inflow into the veins is very free, and where the outflow from the part of the body is not so free, a local venous pulse may develop.

Characters of the Pulse Wave.—If a finger be placed on the carotid artery and another upon the radial artery it will be felt that the artery near the heart expands (pulses) before that further from the heart.

The pulse develops first in the arteries near the heart and passes outwards towards the periphery. The reason for this is obvious. The arteries are always overfilled with blood. The ventricle drives its contents into this overfilled aorta, and to accommodate this the aortic wall expands. But since the aorta communicates with the other arteries this increased pressure passes outwards along them, expanding their wall as it goes.

The pulse wave may thus be compared to a wave at sea, which is also a wave of increased pressure, the only difference being that, while the waves at sea travel freely over the surface, the pulse wave is confined in the column of blood, and manifests itself by expanding the walls of the arteries.

It greatly simplifies the study of the pulse to regard it in this light, and to study it just as we should study a wave at sea.

1. *Velocity.*—To determine how fast a wave is travelling we might select two points at a known distance from one another, and with a watch note how long the wave takes to pass from one to the other. So with the pulse wave, two points on an artery at a known distance from one another may be taken and the time which the wave takes to pass between them may be measured.

It is thus found that the pulse wave travels at about 9 or 10 metres per second—about thirty times as fast as the blood flows in the arteries.

2. *Length of the Wave.*—To determine this in a wave at sea is easy if we know its velocity and know how long it takes to pass any one point. Suppose it is travelling at 50 feet per second, and that it takes one second to pass a particular point, obviously it is 50 feet in length. The same method may be applied to the pulse wave. We know its velocity, and by placing the finger on an artery we may determine that one wave follows another in rapid succession, so that there is no pause between them. Each wave corresponds to a ventricular systole, and therefore each wave must last, at any point, just the time between two ventricular systoles—just the time of a cardiac cycle. There are about 70 cycles per minute—*i.e.* per 60 seconds; hence each must last 0·88 second. The pulse wave takes 0·88 second to pass any place, and it travels at 10 metres per second; its length then is 8·8 metres, or about *five times the length of the body.* It is then an enormously long wave, and it has disappeared at the periphery long before it has finished leaving the aorta.

3. *The Height of the Wave.*—The height of the pulse wave, as of a wave at sea, depends primarily on the pressure causing it, but the character of the arterial wall modifies it very largely. Thus the true height of the pulse wave in the great arteries near the heart is masked by the thickness of the arterial wall.

Speaking generally, however, we may say that the pulse wave is highest near the heart, and becomes lower and lower as it passes out to the periphery, where it finally disappears altogether (Fig. 130). This disappearance is due to its force becoming expended in expanding the arterial wall.

4. *The Form of the Wave.*—Waves at sea vary greatly in form, and the form of the wave might be graphically recorded on some moving surface such as the side of a ship by some floating body. If the ship were stationary a simple vertical line would be produced, but if she were moving a curve would be recorded, more or less abrupt according to her speed. From this curve the shape of the wave might be deduced, if the speed of the vessel is known.

The same method may be applied to the arterial pulse. By recording the changes produced by the pulse wave as it passes any point in an artery the shape of the wave may be deduced.

This may be done by any of the various forms of sphygmograph.

Such a tracing is not a true picture of the wave, but simply of the effect of the wave on one point of the arterial wall. Its apparent length depends upon the rate at which the recording surface is travelling, and not on the length of the wave.

Its height depends in part upon the length of the recording lever, in part upon the resistance offered by the instrument, in part upon the degree of pressure with which the instrument is applied to the artery, and in part on the thickness of the arterial wall.

Such a trace shows (Fig. 131)—

1*st.* That the pulse waves generally follow one another without any interval.

2*nd.* That the rise of the wave is much more abrupt than the fall.

3*rd.* That upon the descent of the primary wave there are one or more secondary waves.

One of these is constant and is very often well marked. It forms a second crest, and is hence called *the dicrotic wave.*

Between the chief crest and this secondary crest, a smaller crest is often manifest (Fig. 131, 3). From its position, it is called *the predicrotic wave.* Sometimes other crests appear.

If the wave has only one crest it is called a one-crested or monocrotic wave. If only the dicrotic crest is well marked it is called dicrotic. If three crests are present, tricrotic; if several crests, polycrotic.

To understand the various parts of the pulse wave it is necessary to compare it with the intra-ventricular pressure changes. This may be done by taking synchronously tracings of the intra-ventricular pressure, and of the aortic pressure (Fig. 132).

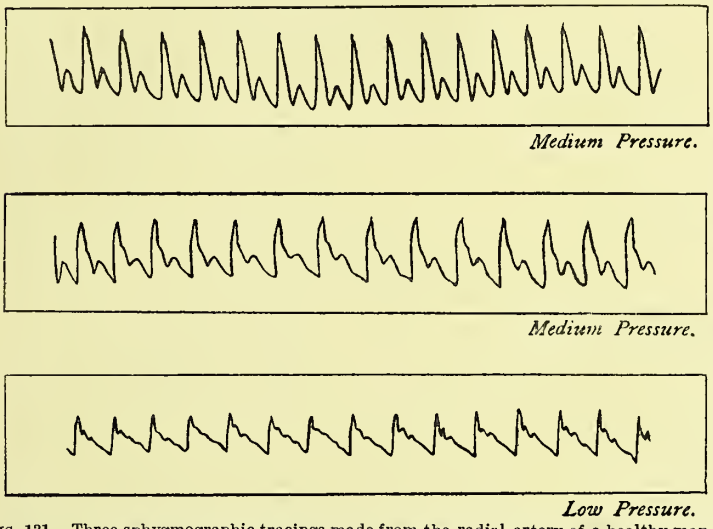


FIG. 131.—Three sphygmographic tracings made from the radial artery of a healthy man in the course of one hour without removing the sphygmograph. 1 was made immediately after muscular exercise; 2 was made after sitting still for half an hour; and 3, after an hour.

Such a tracing shows that at the moment of ventricular systole the pressure in the aorta is higher than that in the left ventricle.

As ventricular systole advances the intra-ventricular pressure rises and becomes higher than the aortic. At that moment the aortic valves are thrown open and a rush of blood takes place into the aorta, raising the pressure and expanding the artery, and causing the up-stroke, and *crest* of the pulse curve. As the ventricle empties itself the intra-ventricular pressure tends somewhat to fall, and, at the same time, a fall in the intra-aortic pressure also takes place. If all the blood does not leave the ventricle in the first gush, *e.g.* when the intra-aortic pressure is high as compared with the force of the heart (Fig. 132, continuous line), there is a residual outflow which arrests the diminution in the aortic pressure, or may actually raise it, causing the *predicrotic wave*. As this residual outflow diminishes, the aortic pressure again falls and continues to fall until the moment of ventricular diastole. At this instant the intra-ventricular pressure suddenly becomes less than the intra-aortic, and the semilunar valves are forced downwards towards the ventricles, and thus the capacity of the aorta is slightly increased and the pressure falls. This fall in pressure is indicated by the *dicrotic notch*. But the elasticity of the semilunar valves at once makes them again spring up, thus increasing the pressure in the aorta and causing the second crest, *the dicrotic wave*. After this the pressure in the arteries steadily diminishes till the mean is reached, to be again increased by the next ventricular systole.

The form of the pulse wave varies according to the relationship between the arterial pressure and the activity of the heart.

If the heart is active and strong in relation to the arterial pressure, the main mass of the blood is expelled in the first sudden outflow, and the residual flow is absent or slight (Fig. 132, dotted line). In this case there is a sudden and marked rise of the arterial pressure, followed by a steady fall till the moment of ventricular diastole. The rebound of the semilunar valves is marked in this case and causes a very prominent dicrotic wave, while the predicrotic wave is absent (Fig. 131, 1). Such a condition is well seen after violent muscular exertion, and in certain fevers. In these conditions the dicrotic wave is so well marked that it can be readily felt with the finger. It is to this form of pulse that the term dicrotic is applied in medicine.

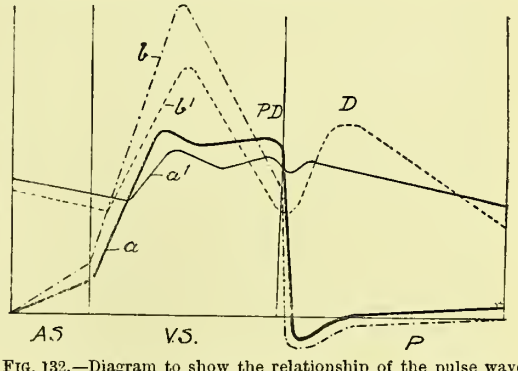


FIG. 132.—Diagram to show the relationship of the pulse wave to the cardiac cycle and the effect of altering the relationship between the activity of the heart and the arterial blood pressure. — · — · — b is the curve of intra-ventricular pressure, and - - - - - b^1 is a pulse curve with an active heart and a relatively low arterial pressure. — a and a^1 are the same with a sluggish heart and a relatively high arterial pressure.

On the other hand, if the ventricles are acting slowly and feebly in relationship to the arterial pressure, the initial outflow of blood does not take place so rapidly and completely (Fig. 132, continuous line), and the initial rise in the pulse is thus not so rapid. The residual outflow of blood is more marked and causes a well-marked

secondary rise in the pulse curve—the predicrotic wave. In certain cases this may be higher than the primary crest, producing the condition known as the anacrotic pulse. The relatively high intra-arterial pressure here prevents the development of a well-marked dicrotic wave.

In extreme cases of this kind, when the arterial walls are very tense, they may recover after their expansion in an irregular jerky manner, and may give rise to a series of katacrotic crests producing a polycrotic pulse (Fig. 131, 3).

From what has been said it will be seen that a study of the pulse wave gives most valuable information as regards the state of the circulation, and the physician constantly makes use of the pulse in diagnosis.

Palpation of Pulse.—On placing the finger on the radial artery the points to determine are—

1*st.* The *rate* of the pulse—*i.e.* the rate of the heart's action.

2*nd.* The *rhythm* of the pulse—*i.e.* of the heart's action as regards—(1) *Strength of the various beats.*—Normally the beats differ little from one another in force—since the various heart beats have much the same strength. Respiration has a slight effect which will afterwards be considered (*see* p. 45). In pathological conditions great differences in the force of succeeding pulse waves occur. (2) *Time relationship of beats.*—Normally the beats follow one another at regular intervals — somewhat shorter during inspiration — somewhat longer during expiration. In pathological conditions great irregularities in this respect may occur.

3*rd.* The *volume* of the pulse wave. Sometimes the wave is high and greatly expands the artery—sometimes less high and expanding the artery less. The former condition is called a full pulse (*pulsus plenus*), the latter a small pulse (*pulsus parvus*). The fulness of the pulse depends upon two factors:—1*st.* The average tension in the arteries between the pulse beats. If this is high, the walls of the artery are already somewhat stretched, and therefore the pulse wave expands them only slightly further. On the other hand, if the average pressure is low, the arterial wall is lax, and is readily stretched to a greater extent. 2*nd.* The force of the heart. To stretch the arterial wall to a large extent requires an actively acting heart throwing a sudden large wave of blood into the arterial system at each systole. The full pulse is well seen after violent exertion, when the heart is active and the peripheral vessels fully dilated, thus allowing a free flow of blood from the arteries and thus keeping the mean arterial pressure low.

4*th. Tension* of the pulse. Sometimes the pulse wave is easily obliterated by pressing on the artery—sometimes considerable force is required to prevent it from passing. To test this, two fingers must be placed on the artery. That placed nearer the heart must be pressed more and more firmly on the vessel until the pulse wave is no longer felt by the second finger. In this way the tension or force of the pulse, the maximum systolic pressure in the artery, may be roughly determined. So important, however, is this point, that various instrumental methods for determining it have been devised (*see* p. 30).

The tension of the pulse varies directly with the force of the heart and with the peripheral resistance. The first statement is so obvious as to require no amplification. It is also clear that if the peripheral resistance is low, so that blood can easily be forced out of the arteries into the capillaries, the arterial wall will not be so forcibly expanded as when the resistance to outflow is great. Hence a high-tension pulse is indicative of a strongly acting heart with constriction of the peripheral vessels. It is well seen during the shivering fit which so frequently precedes a febrile attack, since at that time the peripheral vessels are constricted and the heart's action excited. The tension of the pulse wave must not be confused with the mean arterial pressure (*see* p. 29).

5*th.* The *form* of the pulse wave may be investigated by means of the finger alone or by means of the sphygmograph. The points to be observed are—

(1) Does the wave come up suddenly under the finger? In the *pulsus celer* (or active pulse) it does so; in the *pulsus tardus*, on the other hand, it comes up slowly. The former condition is indicative of an actively acting heart with no great peripheral resistance—the latter indicates that the heart's action is weak in relationship to the arterial blood pressure.

(2) Does the wave fall slowly or rapidly? Normally the fall should not be so sudden as the ascent. When the aortic valves are not properly closed the descent becomes very rapid.

(3) Are there any secondary waves to be observed? The only one of these which can be detected by the finger is the dicrotic wave, and this only when it is well marked. When it can be felt, the pulse is said to be dicrotic, and, as before stated, this indicates an actively acting heart with an arterial pressure low relatively to the strength of the ventricles.

B. *Capillary Pulse*

Normally there is no pulse in the capillaries. Their thin endothelial wall is not well adapted to bear such an intermittent strain. If, however, the arterioles to a district are freely dilated so that little resistance is offered to the escape of blood from the arteries, and if at the same time the outflow from the capillaries is not proportionately increased, intermittent inflow and resistance to outflow are developed, and a pulse

is produced. Such a condition is seen in certain glands during activity.

C. *Venous Pulse*

1. The absence of a general venous pulse has been already explained. But just as in the capillaries so in the veins, a local pulse may develop.

2. In the veins entering the auricles a pulse occurs, but a pulse having no resemblance to

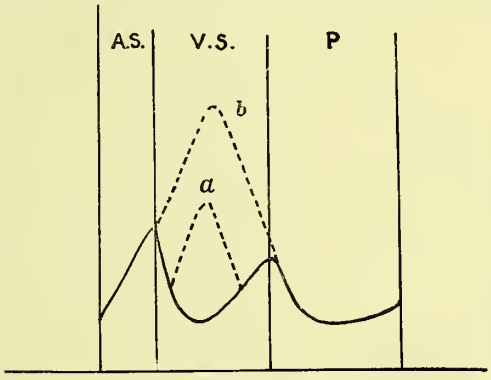


FIG. 133.—Tracings of the pulse in the great veins in relationship to the cardiac cycle. —— normal venous pulse. - - - - *a* and *b* venous pulse in tricuspid incompetence.

the arterial pulse, although depending on the same three factors.

Its form is indicated in Fig. 133.

Its features are to be explained as follows :—

Blood is constantly flowing into the great veins, pressed on from behind. When the auricles contract, the outflow from these veins into the heart is suddenly checked, and consequently the veins distend. At the moment of auricular diastole the outflow is again free, a rush of blood takes place into the distending auricles, and thus the pressure in the veins falls. But as this is occurring, blood is shot from the ventricles into the arteries, and the carotid, lying behind the jugular vein, transmits its pulse to the vein as a crest. While the ventricle is contracted blood cannot pass on from the auricles, and hence it accumulates in the great vein and makes a third crest at the end of ventricular systole. At the moment when the ventricles dilate a sudden rush of blood takes place from the veins and auricles into the ventricles, and thus a sudden fall in the pressure is produced. Gradually, as the ventricles fill, the pressure in the auricles and veins increases and they are again expanded.

This is the normal venous pulse. But, if the auriculo-ventricular valves are incompetent, blood is forced back into the auricles and veins when the ventricles contract, and a crest develops after the carotid crest which it may replace. The height of this crest is a good index of the amount of regurgitation.

Respiratory Variations in Blood Pressure.—Not only do rhythmic changes in the *arterial* pressure occur with each beat of the heart, but larger changes are caused by the respirations—the rise in pressure in great measure corresponding to the phase of inspiration, the fall in pressure to the phase of expiration. This statement is not quite accurate, as will be seen when considering the influence of respiration on circulation (*see* p. 45). These variations are easily seen in a tracing of the arterial pressure taken with the mercurial manometer (Fig. 134).

A pulse synchronous with the respirations may also be observed in the *great veins at the root of the neck* and in the venous sinuses of the cranium when it is opened. With each inspiration they tend to collapse, with each expiration they again expand. The reason for this is that during inspiration the pressure inside the thorax becomes low and hence blood is sucked from the veins into the heart, while during expiration the intra-thoracic pressure becomes higher and thus the entrance of blood into the heart is opposed.

3. MEAN BLOOD PRESSURE

I. PRESSURE IN THE ARTERIES.—A. *Methods.*—The first investigation of the pressure in the blood-vessels was made by the Rev. Stephen Hales in 1733. He fixed a long glass tube in the femoral artery of a horse laid on its back, and found that the pressure supported a column of blood of 8 feet 3 inches, while, when the tube was placed in a vein, only 1 foot was supported.

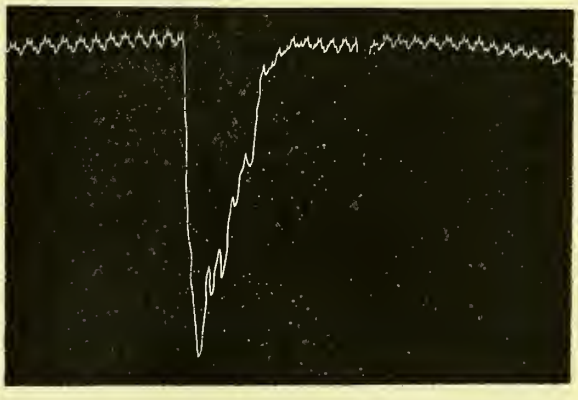
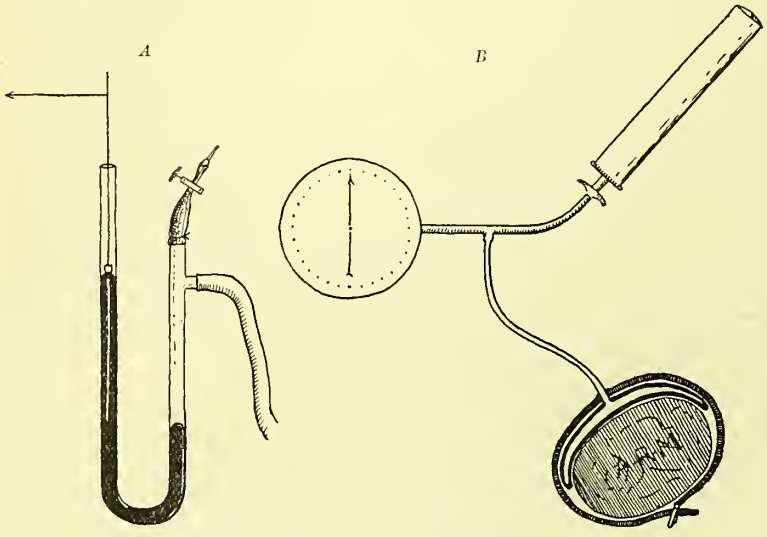


FIG. 134.—Tracing of the arterial blood pressure to show large respiratory variations, and small variations due to heart beats upon these, and the sudden fall in the pressure produced by stimulating the inferior cardiac branch of the vagus nerve.

The capillary pressure is, of course, intermediate between these two.

At the present time, instead of letting the blood pressure act directly against the force of gravity, it is found more convenient, in studying the pressure in an artery, to let it act through a column of mercury placed in a U tube (Fig. 135, *A*). To record the changes in pressure a float is placed upon the mercury

in the distal limb of the tube, and this carries a writing style which records the changes upon a moving surface. With such an apparatus a record such as is shown in Fig. 134 is given. The actual pressure is measured by taking the difference between the level of the mercury in the two limbs of the tube. To make the measurement it is customary to describe an abscissa when the mercury is at the same level in the two sides of the tube. The difference between this and the level of the style at any moment multiplied by two, on account of the depression in the proximal limb which accompanies the elevation in the distal limb, gives the blood pressure in mm. of mercury.



G. 135.—*A*, The mercurial manometer with recording float, used in taking records of the arterial blood pressure of lower animals; *B*, the Hill-Barnard sphygmometer, for measuring the arterial pressure in man.

On the record made with such an instrument the rhythmic variations in the arterial blood pressure already considered on p. 27 are clearly visible.

In the human subject the arterial pressure may be measured by various methods, some of which give the systolic pressure, *i.e.* the pressure at the maximum of the pulse wave; while others give the diastolic pressure—the pressure between the pulse waves. As shown on p. 27 the difference between these is most marked in the great arteries, and falls to zero before the capillaries are reached (Fig. 136).

To measure the systolic pressure it is necessary to find the pressure which must be applied to an artery in order to prevent the pulse from passing.

This may be done with Riva Rocci's apparatus, by applying a bag round the arm so that it rests upon the brachial artery. The bag is firmly strapped on by means of a broad supporting belt, and it is connected with a pump by which the pressure within it may be raised, and with a mercurial manometer by which the pressure applied may be measured in mm. of mercury. The pressure is then raised till the pulse at the wrist is no longer felt, and the column of mercury indicates the systolic pressure in the artery.

Instead of using the radial pulse as the index, the passage of blood into a region rendered bloodless may be used. In Gaërtner's tonometer the bag and band are applied round the finger rendered bloodless at a pressure sufficient to prevent the blood from passing, and then the pressure is slowly lowered till the blood passes and the finger again flushes. The height of the column of mercury at that moment gives the systolic pressure in the small arteries of the finger.

The diastolic pressure may be measured by taking advantage of the fact that when the pressures inside and outside an artery are equal the pulse wave is best marked. A bag and band are applied to the brachial artery, and the movements of the pulse are observed in the column of mercury or on an aneroid barometer in the Hill-Barnard instrument, or by a record taken with a small tambour on a revolving cylinder in Erlanger's instrument (Fig. 137). Erlanger's instrument may also be used as the Riva Rocci for systolic pressures, and it is therefore the most valuable instrument for the study of the arterial pressure in the man.

B. Results.—By these methods it has been found that the systolic pressure in the brachial artery of man is about 110 mm. of mercury, while the diastolic pressure is only about 65 mm. The difference between these, of course, gives the pulse pressure or tension.

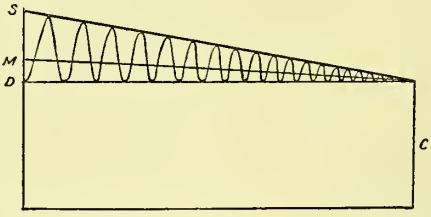


FIG. 136.—To show the difference between systolic, diastolic, and mean blood pressure throughout the arterial system. *S*, Systolic pressure; *D*, diastolic pressure; *M*, mean pressure.

The force of the heart and the degree of peripheral resistance both modify the arterial pressure, and normally these so act together that disturbance of one is compensated for by changes in the other. Thus, if the heart's

action becomes increased and tends to raise the arterial pressure, the peripheral resistance falls and prevents any marked rise. Similarly, if the peripheral resistance is increased, the heart's action is diminished, and no rise in the pressure occurs. Under certain conditions, however, this compensatory action is not complete, and changes in the arterial pressure are thus brought about.

The *volume of blood* has a comparatively small influence on the arterial pressure, because the

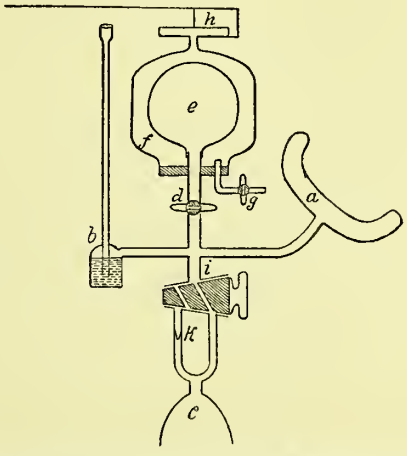


FIG. 137.—Scheme of Erlanger's apparatus for determining the arterial blood pressure in man. *a*, Elastic bag for arm; *b*, mercurial manometer; *c*, pump; *f*, glass bulb; *e*, rubber ball; *h*, tambour with lever.

veins are so large that they accommodate very varying amounts of fluid.

FACTORS CONTROLLING ARTERIAL PRESSURE.—(*a*) *Heart's Action.*—The influence of this may be readily demonstrated by stimulating the vagus nerve while taking a tracing of the arterial pressure. The heart is inhibited, less blood is forced into the arteries, and the pressure falls (Fig 134).

If, on the other hand, the accelerator nerve is stimulated, the increased heart's action drives more blood into the arteries, and the pressure rises.

(*b*) *Peripheral Resistance.*—The resistance to outflow from the arteries depends upon the resistance offered in the small arteries, the walls of which are chiefly composed of visceral muscle fibres. When these fibres are contracted, the lumen of the vessels is small and the resistance is great. When they are relaxed, the lumen of the vessels dilates, and the resistance to outflow is diminished. This muscular tissue of the arterioles acts as a stop-cock to the flow of blood from the arteries to the capillaries. It is of great importance—

1*st.* In maintaining the uniform pressure in the arteries.

2*nd.* In regulating the flow of blood into the capillaries.

During the functional activity of a part, a free supply of blood in its capillaries is required. This is brought about by a relaxation of the muscular coats of the arterioles leading to the part. When the part returns to rest, the free flow of blood is checked by the contraction of the muscular walls of the arterioles.

The action of the arterioles is well seen under the influence of certain drugs (vaso-dilators and vaso-constrictors). If, while a tracing of the arterial pressure is being taken, nitrate of amyl is administered to the animal, it will be seen that the skin and mucous membranes become red and engorged with blood, while at the same time the arterial pressure falls. Nitrites cause the muscular coat of the arterioles to relax, and thus, by diminishing peripheral resistance, permit blood to flow freely from the arteries into the capillaries.

Salts of barium have precisely the opposite effect, causing the skin to become pale from imperfect filling of the capillaries, and producing a marked rise in the arterial pressure. Contraction of the muscles of the arterioles is produced, and the flow of blood from arteries to capillaries is retarded.

Not only is the state of the arterioles influenced thus by drugs, but it is also affected by the internal secretions (*see* p. 82) from certain organs. A powerful vaso-constrictor *adrenalin* is produced in the medulla of the suprarenals.

The condition of the arterioles may be studied in many different ways—

1*st. By direct observation.* — 1. With the naked eye. A red engorged appearance of any part of the body may be due to dilatation of the arteriole leading to it. It may, however, be due to some obstruction to the *outflow* of blood from the part.

2. With the microscope. In certain transparent structures, such as the web of the frog's foot, or the wing of the bat, or the mesentery, it is possible to measure the diameter of the arterioles by means of an eye-piece micrometer, and to study their dilatation and contraction.

2*nd.* The engorgement of the capillaries brought about by dilatation of the arterioles manifests itself also in an increased *size of the part.* Everyone knows how on a hot day, when the arterioles of the skin are dilated, it is difficult to pull on a glove which, on a cold day, when the cutaneous arterioles are contracted, feels loose. By enclosing a part of the body in a case with rigid walls filled with fluid or with air which is connected with some form of recording tambour, an increase or decrease in the size of the part due to the state of its vessels may be registered. Such an instrument is called a plethysmograph.

3*rd.* When the arterioles to a part are dilated and the blood is flowing freely into the capillaries, the part becomes warmer, and by fixing a thermometer to the surface, conclusions as to the condition of the arterioles may be drawn.

4th. By streaming blood through the vessels and observing the rate at which it escapes, the changes in the state of the arterioles may be made out. This perfusion method is much used in studying the action of drugs.

5th. Since the state of the arterioles influences the arterial pressure, *if the heart's action is kept uniform*, changes in the arterial blood pressure indicate changes in the arterioles, a fall of pressure indicating dilatation, a rise of pressure, constriction.

Normal State of Arterioles.—If an arteriole in some transparent tissue be examined, it will be found to maintain a fairly uniform size, but to undergo periodic slow changes in calibre. If the ear of a white rabbit be studied, it will be seen to undergo slow changes, at one time appearing pale and bloodless, at another time red and engorged. During this latter phase numerous vessels appear which in the former condition were invisible. These slow changes are independent of the heart's action and of the rate of respirations. They appear to be due to the periodic rhythmic contraction which is a characteristic property of visceral muscle fibres. This rhythmic action is better marked in some vessels than in others.

Vaso-motor Mechanism.—If the sciatic nerve of a frog be cut, the arterioles in the foot at once dilate. If the sciatic be stimulated, the arterioles become smaller. The same results follow if the anterior roots of the lower spinal nerves, from which the sciatic takes origin, be cut or stimulated.

We must, therefore, conclude (1) that the central nervous system exerts a constant *tonic* influence upon the arterioles, keeping them in a state of semi-contraction; and (2) that this influence may be increased, and thus a constriction of the arterioles caused, and in this way the flow of blood from arteries to capillaries obstructed and the arterial pressure raised; and (3) that this influence may be diminished, so that the arterioles dilate and allow an increased flow into the capillaries from the arteries, and thus lower the arterial pressure.

These mobile arterioles, under the control of the central nervous system, constitute a *vaso-motor mechanism*, which plays a most important part in connection with nearly every vital process in the body. By it the pressure in the arteries is governed, by it the supply of blood to the capillaries and tissues is controlled, and by it the loss of heat from the body is largely regulated (p. 72).

This vaso-motor mechanism consists of the three parts:—

1*st.* The contractile muscular walls of the arterioles with the nerve terminations in them.

2*nd.* The nerves which pass to them.

3*rd.* The portions of the central nervous system presiding over these.

1. *Muscular Walls of the Arterioles.*—The muscular fibres are maintained in a state of tonic semi-contraction by nerves passing to them, and when these nerves are divided, the muscular fibres relax. But if, after these nerves have been cut, the animal be allowed to live, in a few days the *arterioles again pass into a state of tonic semi-contraction*, although no union of the divided nerve has taken place.

Certain drugs, *e.g.* digitalis and the salts of barium, act as direct stimulants to these muscle fibres.

It appears that the *muscular fibres* in the arterioles, as elsewhere, tend to maintain themselves in a state of partial contraction, which increases and diminishes in a regular rhythmic manner.

The precise part played by the nerve terminations has not been definitely established, but certain drugs appear to act specially upon them. Thus apocodeine, while it does not prevent barium salts from constricting the vessels, prevents the constricting action of extracts of the medulla of the suprarenals, even when the nerves are cut. Hence it must be concluded that it paralyses a nervous mechanism in the arteriole wall which is stimulated by the suprarenal extract.

Normally this muscular mechanism is controlled by the nervous system.

2. *Vaso-motor Nerves.*—When a nerve going to any part of the body is cut the arterioles of the part generally dilate, when it is stimulated the arterioles are *usually* contracted; sometimes, however, they are dilated. In no case does section of a nerve cause constriction of the arterioles.

These facts prove that the vaso-motor nerves may be divided into two classes:—

1*st.* Vaso-constrictor.

2*nd.* Vaso-dilator.

A. Vaso-constrictor Nerves.—The fact that section of these at once causes a dilatation of the arterioles proves that they are constantly transmitting impulses from the central nervous system.

Course.—The course of these fibres has been investigated by section and by stimulation (Fig. 138).

They leave the spinal cord chiefly in the dorsal region by the anterior roots of the spinal nerves, pass into the sympathetic ganglia, where they have their cell stations, and then as non-medullated fibres pass, either along the various sympathetic nerves to the viscera, or back through the grey ramus (*see* Fig. 83, vol. vii. p. 550), into the spinal nerve, and run in it to their terminations.

B. Vaso-dilator Nerves.—A good example of such a nerve is to be found in the chorda tympani branch of the facial nerve, which sends fibres to the submaxillary and sublingual salivary glands. If this nerve be cut, no change takes place in the vessels of the gland, but

when it is stimulated the arterioles dilate and allow an increased flow of blood through the capillaries. These fibres, therefore, instead of increasing the activity of muscular contraction, diminish or inhibit it. They play the same part in regard to the muscular fibres of the arterioles as the inferior cardiac branch of the vagus does in regard to the cardiac muscular fibres. As examples of vaso-dilator nerves, the gastric branches of the vagus carrying vaso-dilator fibres to the mucous membrane of the

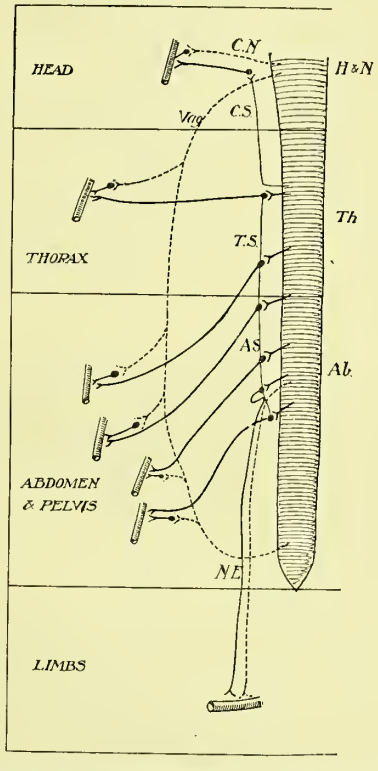


FIG. 138.—Diagram of the distribution of vaso-motor nerves. The continuous line shows the vaso-constrictors, the dotted line the vaso-dilators. *C.N.*, Cranial nerves; *Vag.*, vagus; *T.S.*, thoracic sympathetic; *A.S.*, abdominal sympathetic; *N.L.*, nerves to the leg.

stomach, and the nervi erigentes carrying vaso-dilator fibres to the external genitals, may be taken.

The vaso-dilator nerves of most parts of the body run side by side with the vaso-constrictor nerves; and, hence, curious results are often obtained. If the sciatic nerve of a dog be cut, the arterioles of the foot dilate. If the peripheral end of the cut nerve is stimulated, the vessels contract. But, after a few days, if the nerve be prevented from uniting, the arterioles of the foot recover their tonic contraction, and, if the sciatic nerve is then stimulated, a dilatation, and not a constriction, is brought about. The vaso-constrictor fibres seem to die more rapidly than the vaso-dilator fibres which run alongside of them. Under certain conditions the activity of the vaso-dilator fibres seems to be increased. Thus, if, when the limb is warm, the sciatic nerve is stimulated, dilatation rather than constriction may occur. Again, while rapidly repeated and strong induction shocks are apt to cause constriction, slower and weaker stimuli tend to produce dilatation.

Course. — The vaso-dilator nerves pass out chiefly by the anterior roots of the various spinal nerves, and do not pass through the sympathetic ganglia, but run as medullated fibres to their terminal ganglia (Fig. 138). Bayliss has recently shown that the vaso-dilator fibres for the hind limb leave the cord by the posterior roots, and that they are connected with the cells in the ganglia.

3. *Portions of Nervous System Presiding over the Vaso-motor Mechanism.* — Since a set of nerves causing constriction of the arterioles, and another set causing dilatation exist, we must conclude that there are two mechanisms in the central nervous system—one a vaso-constrictor, the other a vaso-dilator.

A. VASO-CONSTRICTOR CENTRE.—(*a*) *Mode of Action.*—This mechanism is constantly in action, maintaining the tonic contraction of the arterioles.

If any afferent nerve be stimulated, the effect is to increase the activity of the mechanism, to cause a general constriction of arterioles, and thus to raise the general arterial pressure. The centre is, therefore, capable of reflex excitation. In ordinary conditions so many afferent nerves are constantly being stimulated, that it is not easy to say how far the tonic action of this centre is reflex and dependent on the stream of afferent impulses.

But this centre may also be directly acted upon by the condition of the blood and lymph circulating through it. When the blood is not properly oxygenated and freed from carbon dioxide, as in asphyxia or suffocation, this centre is stimulated and a general constriction of arterioles with high blood pressure results.

(*b*) *Position.*—In investigating the position of the centre we may take advantage of—

1*st.* Its constant tonic influence. Removal of the centre at once causes dilatation of arterioles.

2*nd.* The fact that it may be reflexly stimulated. If the vaso-constrictor centre be removed, stimulation of an afferent nerve no longer causes constriction of the arterioles.

Removal of the whole brain above the pons Varolii leaves the centre intact.

Separation of the pons Varolii and medulla oblongata from the spinal cord at once causes a dilatation of the arterioles of the body, and prevents the production of reflex constriction by stimulation of an afferent nerve.

The main part, at least, of the vaso-constrictor mechanism is therefore situated in the pons Varolii and medulla oblongata.

The extent of this centre has been determined

by slicing away this part of the brain from above downwards, and studying the influence of reflex stimulation after the removal of each slice.

It is found that at a short distance below the corpora quadrigemina, the removal of each succeeding part is followed by a diminution in the reflex constriction, until, at a point close to and just above the calamus scriptorius, all reflex response to stimulation stops.

The centre is therefore one of very considerable longitudinal extent.

But it has been found that if, after section of the spinal cord high up, the animal be kept alive for some days, the dilated arterioles again contract, and stimulation of afferent nerves entering the cord below the point of section causes a further constriction. If now another section be made further down the cord, the arterioles supplied by nerves coming from below the point of section will again dilate. This shows that secondary vaso-constrictor centres, tonic in action and capable of having their activity reflexly increased, exist all down the grey matter of the spinal cord. Normally these are under the domain of the dominant centre, but when this is out of action they then come into play.

B. Vaso-dilator Centre.—(*a*) *Mode of Action.*—This mechanism is not constantly in action. Section of a vaso-dilator nerve does not cause vascular dilatation. It may be excited reflexly, but in a different manner from the vaso-constrictor mechanism.

Stimulation of an afferent nerve causes a dilatation of the arterioles in the part from which it comes, and a constriction of the arterioles throughout the rest of the body. If a sapid substance, such as pepper, be put in the mouth, the buccal mucous membrane and the salivary glands become engorged, while there is a constriction of the arterioles throughout the body. The vaso-dilator central mechanism is not general in its action like the vaso-constrictor, but is specially related to the different parts of the body.

This is a matter of the greatest importance in physiology and pathology. It explains the increased vascularity of a part when active growth is going on. The changes in the part, or the products of these, stimulate the afferent nerve. This reflexly stimulates the vaso-dilator mechanism of the part, and thus causes a free flow of blood into the capillaries, and at the same time maintains or actually raises the arterial pressure by causing a general constriction of the arterioles, and thus forces more blood to the situation in which it is required.

The same process occurs in the case of the stomach during digestion, in the case of the kidney during secretion, and in the process of inflammation.

Not only does peripheral stimulation act in this way, but various states of the brain, accompanied by emotions, may stimulate part of the vaso-dilator mechanism, as in the act of blushing.

Again, it has been shown that stimulation of the central end of the depressor nerve (superior cardiac branch of the vagus) causes a dilatation of the arterioles chiefly in the abdominal cavity, but also throughout the body generally. This is the most generalised vaso-dilator reflex known (*see* p. 23).

(*b*) *Position.* — While the dominant vaso-constrictor centre is in the medulla, the vaso-dilator centres seem to be distributed throughout the medulla and spinal cord.

II. Pressure in the Capillaries.—This may be determined by finding the pressure required to blanch the skin or to occlude the capillaries of some transparent membrane.

It has already been shown that the pressure is less than in the arteries and greater than in the veins.

Like the pressure in the arteries, it depends upon the two factors—

1*st.* Force of inflow.

2*nd.* Resistance to outflow.

1*st. Variations in the Force of Inflow.*—The capillary pressure may undergo marked local changes through the *vaso-motor mechanism.* Wherever the function of a part is active, dilatation of the arterioles and an increased capillary pressure exists, and when the influence of vaso-dilator nerves is withdrawn, the capillary pressure falls.

But the capillary pressure may also be modified by the *heart's action*, inasmuch as the arterial pressure, by which blood is driven into the capillaries, depends upon this. In cardiac inhibition not only is arterial pressure lowered, but capillary pressure may also fall. In augmented heart action both arterial and capillary pressure are raised.

2*nd. Variations in Resistance to Outflow.*—Normally the flow from capillaries to veins is free and unobstructed; but, if the veins get blocked, or if the flow in them is retarded by gravity, the capillaries get engorged with the blood which cannot escape from them. This increased pressure in the capillaries is very different from that caused by increased inflow. The flow through the vessels is slowed or may be stopped instead of being accelerated, and the blood gets deprived of its nourishing constituents, loaded with waste products, and tends to exude into the lymph spaces, causing *dropsy.*

It is, therefore, most important to distinguish between high capillary pressure from dilated arterioles or an active heart, and high pressure due to venous obstruction.

A condition very similar to that described, but producing a *capillary pressure high relatively to the pressure in the arteries—though not absolutely*

high—is seen in cases of failure of the heart, when that organ is not acting sufficiently strongly to pass the blood on from the venous into the arterial system. Here the arterial pressure becomes lower and lower, the venous pressure higher and higher, and along with this the capillary pressure becomes high in relationship to the arterial pressure. The blood is not forced through these channels, and congestion of the capillaries and dropsy may result.

The *influence of gravity* plays a very important part on the capillary pressure, since it has so marked an influence on the flow of blood in the veins. When, through heart failure, the blood is not properly sucked up from the inferior extremities, this increased pressure becomes very marked indeed.

3rd. But the pressure in the capillaries may also to a certain extent be varied by the *withdrawal of water from the body*, as in purgation or diuresis, or by the addition of large quantities of fluid to the blood. The venous system is,

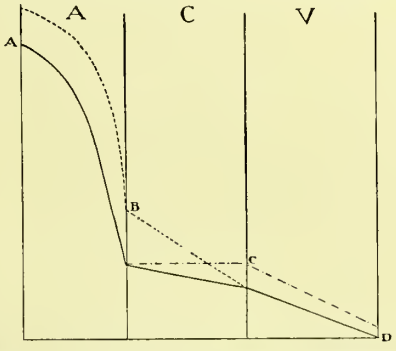


FIG. 139.—The changes in blood pressure in the capillaries produced by increasing the arterial pressure - - - - - -, and by obstructing the venous flow — · — · —. *A*, Arteries; *C*, capillaries; *V*, veins.

however, so capacious that very great changes in the amount of blood in the vessels may take place without materially modifying the arterial or capillary pressure while affecting temporarily the venous pressure.

III. PRESSURE IN THE VEINS.—The pressure in the veins is so low that it may best be determined by a water manometer.

In the veins the force of inflow is small; the resistance to outflow is nil. Hence the pressure is small, and steadily diminishes from the small veins to the large veins entering the heart (Fig. 139).

The venous pressure may be modified by variations in these two factors. Constriction of the arterioles tends to lower the venous pressure, dilatation to raise it. On the other hand, increased heart's action, which so markedly tends to raise arterial pressure, diminishes the pressure in the larger veins, because the blood is thus more rapidly driven from veins into arteries, and because the heart, which in its powerful systole drives out more blood, in its diastole sucks in more.

Compression of the thorax has a very marked effect in retarding the flow of blood from the great veins into the heart, and thus tends to raise the venous pressure and to lower the arterial pressure. Venous pressure may be temporarily modified by the *loss or gain of water*.

IV. PRESSURE IN THE LYMPHATICS.—No exact determination of the lymph pressure in the tissue spaces has been made, but since there is a constant flow from these spaces through the lymphatic vessels and through the thoracic duct into the veins at the root of the neck, the pressure in the tissue spaces must be higher than the pressure in the great veins.

This pressure is kept up by the formation of lymph from the blood, and from the cells of the tissues (*see* p. 10).

B.—FLOW OF BLOOD

The flow of blood, as already indicated, depends upon the distribution of pressure, a fluid always tending to flow from the point of higher pressure to the point of lower pressure. Since a high pressure is maintained in the aorta and a low pressure in the veins entering the heart and in the cavities of the heart during its diastole, the blood must flow through the circulation from arteries to veins.

Velocity.—The velocity of the flow of a fluid depends upon the width of the channel. Since in unit of time unit of volume must pass each point in a stream if the fluid is not to accumulate at one point, the velocity must vary with the sectional area of the channel. In the case of a river, in each second the same amount of water must pass through the narrowest and through the widest part of its channel. Now for a ton of water to get through any point in a channel one square yard in sectional area in the same time as it takes to pass a point in a channel ten square yards in area, it must obviously flow with greater velocity. This may be stated in the proposition that the velocity (V) of the stream is equal to the amount of blood passing any point per second (v) divided by the sectional area of the stream (S)—

$$V = \frac{v}{S}$$

where S is the radius squared multiplied by the constant 3·14.

In the vascular system the sectional area of the aorta is small when compared with the sectional area of the smaller arteries; while the sectional area of the capillary system is no less than 700 times greater than that of the aorta. In the venous system the sectional area steadily diminishes, although it never becomes so small as in the corresponding arteries, and where the great veins enter the heart it is about twice the sectional area of the aorta (Fig. 140).

This arrangement of the sectional area of the

stream gives rise to a rapid flow in the arteries, a somewhat slower flow in the veins, and to a very slow flow in the capillaries.

The suddenness of the change of pressure has a certain influence on the rapidity of flow, as is well seen in a river. If the water descends over a sudden declivity to a lower level it

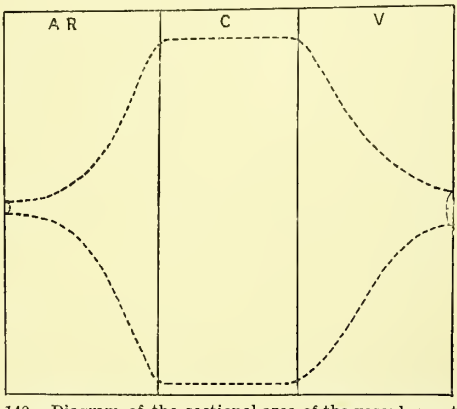


FIG. 140.—Diagram of the sectional area of the vascular system, upon which the velocity of the flow depends. *AR.*, Arteries; *C.*, capillaries; *V.*, veins.

attains a much greater velocity than if the declivity is gentle. In the first case the change of pressure is sudden, in the second case it is slow.

Hence, if, from any cause, the pressure is raised at any point, the flow will tend to be more rapid from that point onwards till the normal distribution of pressure is re-established.

Friction has also a certain effect. A river runs much faster in mid-stream than along the margins, because near the banks the flow is delayed by friction, and the more broken up and subdivided is the channel, the greater is the friction and the more is the stream slowed.

When, therefore, in the capillary system the blood stream is distributed through innumerable small channels, the friction is very great, and this tends to dam back the blood.

The velocity of flow in the arteries and veins may be measured by various methods, of which one of the best is that by means of the *stromuhr*, an instrument by which the volume of blood passing a given point in an artery or vein in a given time may be determined.

The velocity of the flow in the capillaries may be measured in transparent structures by means of a microscope with an eye-piece micrometer. The velocity of the blood is—

Carotid of the dog about	300 mm.	per sec.
Capillaries about	0·5 to 1 m.	,, ,,
Vein (jugular) about	150 mm.	,, ,,

It is not so easy to give definite figures for the velocity of the lymph stream.

Disturbance of any of the factors which govern the rate of flow will bring about alterations in the velocity of the blood in arteries, capillaries, and veins. Thus, an increased venous pressure by leading to a diminution in the difference of pressure, between arteries and veins, will materially slow the blood stream. Great dilatation of the arterioles will slow the blood stream in them; and increased viscosity of the blood, by increasing friction with the vessel wall, will also slow the stream.

SPECIAL CHARACTERS OF BLOOD FLOW.—(*a*) *Arteries.*—The flow of blood in an artery is rhythmically accelerated with each ventricular systole. This is due to the pulse wave. As the wave of high pressure passes along the vessels, the blood tends to flow forwards and backwards from it—so that in front of the wave there is an acceleration of the stream, and behind it a retardation. In a wave at sea the same thing happens, and a cork floating on the surface is moved forward in front of the wave, and again backwards after the wave has passed.

(*b*) *Capillaries.*—In the capillaries the flow is uniform.

(*c*) *Veins.*—In most veins, too, it is uniform, but in the great veins near the heart it undergoes accelerations—

1*st.* With each diastole of auricle and ventricle.

2*nd.* With each inspiration.

3*rd.* By muscular action squeezing the blood out of the small veins.

In all vessels the blood in the centre of the stream moves more rapidly than that at the periphery on account of the friction between the blood and the vessels. An "*axial*" rapid and "*peripheral*" slow stream are, therefore, described. This is well seen in any small vessel placed under the microscope, and in such situations it will be found that, while the erythrocytes are chiefly carried in the axial stream, the leucocytes are more confined to the peripheral stream, where they may be observed to roll along the vessel wall with a tendency to adhere to it.

When from any cause the flow through the capillaries is brought to a standstill, the leucocytes creep out through the vessel wall and invade the tissue spaces. This is the process of *diapedesis*, which plays an important part in inflammation.

Special characters of the Circulation in certain situations

1. *Circulation inside the Cranium* (Fig. 141).—Here the blood circulates in a closed cavity with rigid walls, and therefore its amount can vary only at the expense of the cerebro-spinal fluid. This is small in amount, and permits of very small variations in the volume of blood. Increased arterial pressure in the body does not therefore increase the amount of blood in the brain, but simply drives the blood more rapidly through the organ. There seems to be no regulating nervous mechanism connected with

the arterioles of the brain, and the cerebral pressure simply follows the changes in the general arterial pressure. The splanchnic area is the great regulator of the supply of blood to the brain. Since the cerebral arteries are supported and prevented from distending by the solid wall of the skull, the arterial pulse tends to be propagated into the veins. In these veins the respiratory pulse also is very well marked.

2. *Circulation in the Lungs.* — Vaso-motor nerves seem to be absent, and hence drugs like

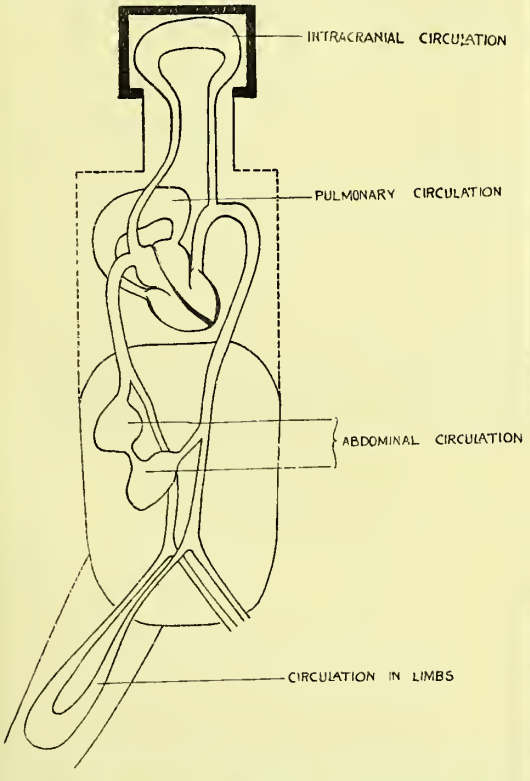


FIG. 141.—Scheme of the circulation, modified from Hill, to illustrate the influence of the various extra-cardiac factors which maintain the flow of blood.

adrenalin fail to cause a constriction of the arterioles. The amount of blood in the lungs is regulated by the blood pressure in the systemic vessels.

3. *Circulation in the Heart Wall.*—A peripheral vaso-motor mechanism is not present in the arterioles of the coronary vessels (*see also* p. 18).

4. *Circulation in the Spleen.* — Here the blood has to flow through a labyrinth of spaces in the spleen pulp, and it is driven on by the alternate contraction and relaxation of the non-striped muscles in the capsule and trabeculæ (*see* p. 9).

Extra-cardiac factors maintaining Circulation

In considering the flow of blood through the vessels due to the distribution of pressure in arteries and veins, it must be remembered that the central pump or heart is not the only factor maintaining it (Fig. 141).

The thorax in the movements of respiration is a suction pump of considerable power, which draws blood into the heart during inspiration. While the auricles may be regarded as the *cisterns* of the heart, the abdominal blood-vessels are the great blood *reservoir*, and the diaphragm contracting in inspiration presses the blood from this reservoir up into the thorax and heart. When, in intermittent muscular exercise, the abdominal muscles are tightened and the respiratory movements of the thorax are increased in the panting which accompanies it, the blood is partly pressed and partly sucked from the abdomen into the heart, and so forced on into the arteries. Even expiration helps in this, for the blood which has filled the vessels of the lungs in inspiration is driven on into the left side of the heart in expiration. The blood is thus forced on into the arteries, and so to the muscles, and they, by their alternate contraction and relaxation, further help to drive it on to the veins, where the valves prevent any back flow during relaxation and thus accelerate the circulation. The high arterial tension thus produced tends to drive the blood through the cranial vessels. The benefit of intermittent muscular exercise on the circulation is thus manifest.

When, on the other hand, some sustained muscular strain has to be undergone, the thorax is fixed, and hence (1*st*) the pressure on the heart and thorax organs is raised, and the increased pressure in the thorax helps to support the heart and to prevent over-distension. The abdominal vessels are also pressed upon, and the sustained contraction of the limb muscles tends to prevent the blood flowing through them. It is thus forced to the central nervous system in which the pressure rises, and if a weak spot in the vessels is present, rupture is apt to occur. (2*nd*) But, if the effort is still further sustained, the high intra-thoracic pressure tends to prevent proper diastolic filling of the heart; blood is therefore not sent on from the veins into the arteries, the veins become congested and the arterial pressure falls, less blood goes to the brain, and thus fainting may result.

In the "head down position," the accumulation of blood in the dependent parts is prevented in the head by the vessels being packed inside the skull, and in the right side of the heart by the supporting pericardium.

In man the position of the abdominal reservoir of blood at a lower level than the heart increases the work of that organ. For this reason, in people with a weak heart, the sudden assumption of the erect position may lead to a failure of the heart and to fainting. Especially is this the case when the abdominal wall is lax, so that accumulation of blood in the abdominal

vessels is not prevented. In the recumbent position, when the reservoir is on the same level as the pump, the work is much easier.

Fainting is a sudden loss of consciousness produced by failure in the supply of blood to the brain. It is accompanied by loss of control over the muscles, so that the individual falls to the ground. It may be induced by any sudden lowering of the arterial blood pressure, whether due to decreased inflow of blood or to decreased peripheral resistance.

Decreased inflow may be caused by—(*a*) Cardiac inhibition brought about reflexly (1) by strong stimulation of ingoing nerves, and more especially of the nerves of the abdomen; (2) by strong stimulation of the upper brain neurons, accompanied by changes in the consciousness of the nature of emotions;—(*b*) Failure of the heart to pump blood from veins to arteries against the force of gravity, as when the erect position is suddenly assumed by people with weak hearts.

Decreased resistance to outflow through sudden dilatation of arterioles may result from changes in the upper brain neurons, accompanied by emotional states and also in digestive disturbances.

However induced, the anæmic state of the brain leads to a stimulation of the cardiac inhibitory centre, and the condition is thus accentuated. The cerebral anæmia is accompanied by pallor of the face.

The treatment consists in depressing the head to allow the force of gravity to act in filling the cerebral vessels and in giving diffusible stimulants to increase the action of the heart.

The Time taken by the Circulation

This was first determined by injecting ferrocyanide of potassium into the proximal end of a cut vein, and finding how long it took to appear in the blood flowing from the distal end. From observation in the horse, dog, and rabbit, it appears that the time corresponds to about twenty-seven beats of the heart, so that in man it should amount to about twenty-three seconds.

Stewart has investigated the rate of flow through different organs by injecting salt solution into the artery, and by detecting its appearance in the vein by the change in the electric conductivity of the contents of the vessel.

Section VI. — Supply of Nourishing Material to the Blood and Lymph, and Elimination of Waste Matter from them.

I. Respiration.—If an animal be placed in a closed chamber filled with ordinary atmospheric air which contains by volume seventy-nine parts of nitrogen and twenty-one parts of oxygen, and if the air be examined after a time, it will be found that the oxygen has diminished in amount, and that a nearly corresponding amount of carbon dioxide has been added.

The same thing occurs in aquatic animals—the water round them loses oxygen and gains carbon dioxide. An animal takes up oxygen and gives off carbon dioxide. This is the process of external respiration.

A. External Respiration

I. *Respiratory Mechanism.*—In aquatic animals the mechanism by which this process is carried on is a gill or gills. Each consists of a process from the surface covered by a very thin layer of integument, just below which is a tuft of capillary blood-vessels. The oxygen passes from the water to the blood; the carbon dioxide from the blood to the water.

A lung is simply a gill or mass of gills, turned outside in, with air instead of water outside the integument. While in aquatic gill-bearing animals there is constantly a fresh supply of water passing over the gills, in lung-bearing animals the air in the lung sacs must be exchanged by some mechanical contrivance.

The lungs consist of myriads of small thin-walled sacs attached round the funnel-like expansions in which the air-passages (infundibular passages) terminate.

Each sac is lined by a layer of simple squamous epithelium, supported by a framework of elastic fibrous tissue richly supplied with blood-vessels. It has been calculated that, if all the

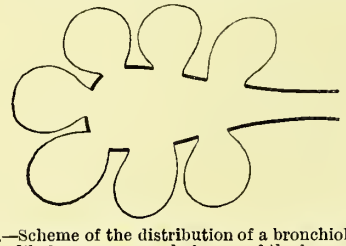


Fig. 142.—Scheme of the distribution of a bronchiole, infundibular passage, and air sacs of the lung.

air vesicles in the lungs of a man were spread out in one continuous sheet, a surface of about 100 square metres would be produced, and that the blood capillaries would occupy about 75 square metres of this. Through these vessels about 5000 litres of blood would pass in twenty-four hours.

The larger air-passages are supported by pieces of hyaline cartilage in their walls, but the smaller terminal passages, the bronchioles, are without this support, and are surrounded by a specially well-developed circular band of non-striped muscle — the bronchial muscle— which governs the admission of air to the infundibula and air sacs.

The lungs are packed in the thorax round the heart, completely filling the cavity.

They may be regarded as two compound *elastic-walled* sacs, which completely fill an air-tight box with movable walls—the thorax—and communicate with the exterior by the windpipe or trachea.

No air exists between the lungs and the sides and base of the thorax, so that the so-called pleural cavity is simply a potential space. If the thoracic wall be punctured so that this potential pleural cavity is brought into connection with the air, the lungs immediately collapse and occupy a small space posteriorly round the large bronchi. This is due to their elasticity (Fig. 143).

The lungs are kept in the distended condition in the thoracic cavity by the atmospheric pressure within them.

Their elasticity varies according to whether the organs are stretched or not. As they collapse, their elastic force naturally becomes less and less; as they are expanded, greater and

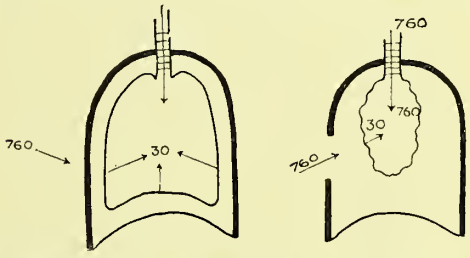


FIG. 143.—Shows the distribution of pressure in the thorax with the chest wall intact, and with an opening into the pleural cavity. (↓) indicates the atmospheric pressure of 760 mm. of mercury; 30 is the elasticity of the lungs also in mm. of mercury.

greater. Taken in the average condition of expansion in which they exist in the chest, the elasticity of the excised lungs of a man is capable of supporting a column of mercury of about 30 mm. in height, so that they are constantly tending to collapse with this force.

But the *inside* of the lungs freely communicates with the atmosphere, and this, at the sea level, has a pressure of about 760 mm. Hg. During one part of respiration this pressure becomes a few mm. less, during another part a few mm. more; but the mean pressure of 760 mm. of mercury is constantly expanding the lung, and acting against a pressure of only 30 mm. of mercury, tending to collapse the lung.

Obviously, therefore, the lungs must be kept expanded and in contact with the chest wall.

When a pleural cavity is opened, the distribution of forces is altered, for now the atmospheric pressure tells also on the outside as well as on the inside of the lungs and acts along with the elasticity of the organ. So that now a force of 760 mm. + 30 mm. = 790 mm. acts against 760 mm., causing a collapse of the lungs.

In the surgery of the thorax, as well as in the physiology of respiration, these points are of great importance.

II. *Physiology.* — The process of external respiration consists of two parts—

1*st.* The passage of air into and out of the air sacs.

2*nd.* The interchange of gases between the air in the air vesicles and the blood in the capillaries.

A. *Passage of Air into and out of the Lungs*

This is brought about—

1*st.* By the movements of respiration—breathing.

2*nd.* By diffusion of gases.

The air is made to pass into and out of the lungs by alternate *inspiration* and *expiration.*

1. *Movements of Respiration—A. Inspiration.*—During this act the thoracic cavity is increased in all directions—lateral, antero-posterior, and vertical. As the thorax expands, the air pressure inside the lungs keeps them pressed against the chest wall, and the lungs expand with the chest. As a result of this expansion of the lungs the pressure inside becomes less than the atmospheric pressure, and air rushes in until the pressure inside and outside again becomes equal. This can be shown by placing a tube in the mouth or in a nostril and connecting it with a water manometer.

This expansion of the lungs can readily be determined in the vertical direction by percussion, and in the horizontal planes by measurement. By tapping the chest with the finger over the lung in the right intercostal spaces, a resonant note is produced, while if the percussion is performed below the level of the lung, a dull note is heard. If the lower edge of this resonance be determined before an inspiration, and again during it, it will be found to have descended.

As a result of inspiration, the form of the chest is markedly modified, the change being best seen in transverse sections. In expiration the chest in transverse section is an elongated ellipse from side to side, in inspiration it becomes more circular (Fig. 144). The change from side to side and from behind forwards is best marked towards the lower part of the chest, less marked in the upper part. These changes may be recorded by means of a Cyrtometer, a piece of flexible gas-tubing hinged behind, so that it can be modelled to the chest.

The change from above downwards cannot be directly seen, but it is indicated by a forward movement of the wall of the abdomen. It will be described when considering the mechanism by which it is brought about.

The expansion of the chest in inspiration is a muscular act and is carried out against the following forces—

1*st. The Elasticity of the Lungs.*—To expand the lungs their elastic force has to be overcome, and the more they are expanded the greater is their elasticity. This factor therefore plays a smaller part at the beginning than towards the end of inspiration.

2*nd. The Elasticity of the Chest Wall.*—The

resting position of the chest is that of expiration. To expand the chest the costal cartilages have to be twisted.

3rd. The Elasticity of the Abdominal Wall.—As the cavity of the thorax increases downwards, the abdominal viscera are pushed against the muscular abdominal wall, which, in virtue of its elasticity, resists the stretching force.

In studying how these changes are brought about we may consider—

1st. The Increase in the Thorax from above downwards.

This is due to the contraction of the diaphragm (Fig. 144).

In expiration this dome-like muscle, rising from the vertebral column and from the lower costal margin, arches upwards, lying for some distance along the inner surface of the ribs and then curving inwards to be inserted into the flattened central tendon, to which is attached the pericardium and on which rests the heart.

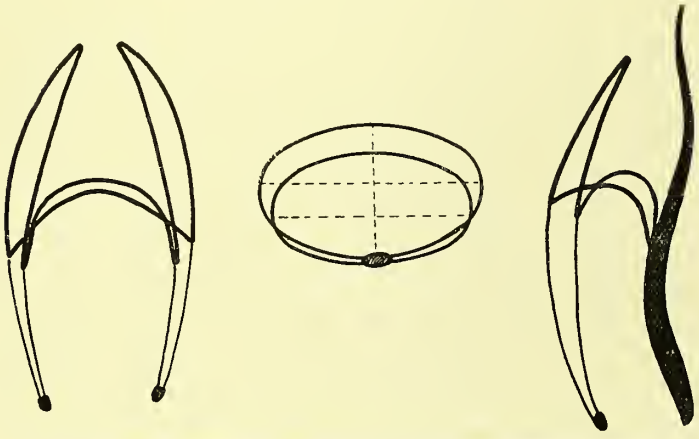


FIG. 144.—Vertical-tangential, transverse, and vertical-mesial sections of the thorax in inspiration and expiration.

In inspiration the muscular fibres contract. But the central tendon being fixed by the pericardium does not undergo extensive movement. The result of the muscular contraction is thus to flatten out the more marginal part of the muscle and to withdraw it more or less from the chest wall—thus opening up a space, the complemental pleura, into which the lungs expand (Fig. 144).

The existence of this complemental pleura is of great importance in cases of accumulation of fluid in the chest, for the fluid may collect and push down the diaphragm without materially altering the lower margin of the lungs.

It might be expected that this contraction of the diaphragm would pull inwards the chest wall—but this is prevented by the expansion of the thorax in the lateral and antero-posterior diameters as a result of the mechanism which has next to be considered. Nevertheless in young children, in whom the chest wall is soft, an indrawing of the chest with each contraction of the diaphragm may occur, and may lead to permanent distortion of the thorax.

2nd. The Increase in the Chest in the antero-posterior and lateral diameters.

This is brought about by the elevation of the ribs, which rotate round the axis of their attachments to the vertebral column.

To understand this, the mode of the connection of the ribs to the vertebral column must be borne in mind. The head of the rib is attached to the bodies of two adjacent vertebræ. The tubercle of the rib is attached to the transverse process of the lower of these vertebræ. From this the shaft of the rib projects outwards, downwards and forwards, to be attached in front to the sternum by the costal cartilage. If the rib is made to rotate round its two points of attachment, its lateral margin is elevated and carried outwards, while its anterior end is carried forwards and upwards (*see* Fig. 145).

Further, as we pass from above downwards, each pair of ribs forms the arc of a larger and larger circle, and as each pair rises it takes the place of a smaller pair above. In these ways, the chest is increased from before backwards and from side to side.

The first pair of ribs does not undergo this movement; the motion of the second pair of ribs is slight, but the range of movement becomes greater and greater as we pass downwards until the floating ribs are reached, and these are fixed by the abdominal muscles. This greater movement is simply due to the greater length of the muscles moving the ribs. The muscles are chiefly the external intercostal muscles, and these may be considered as acting from the fixed first rib. Now, if the fibres of the first intercostal muscle are one inch in length, the second rib can be raised, say, half an inch. The first and second intercostals acting on the third rib will together be two inches in length, and in contracting they can raise the third rib through, say, half of two inches—*i.e.* one inch. The first, second, and third intercostals, acting on the fourth rib, are three inches in length, and can therefore raise this rib half of three, or one

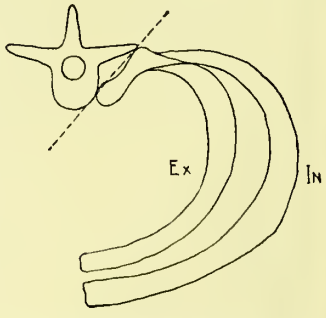


FIG. 145.—Shows the movements of the ribs from their position in expiration to their position in inspiration.

and a half inches, and so through the other ribs, until the floating ribs fixed by the abdominal muscles are reached.

When the diaphragm takes the chief part in inspiration the breathing is said to be abdominal in type—when the intercostals chiefly act in raising the ribs it is said to be thoracic. Abdominal breathing is best marked in males—thoracic in females.

Along with the intercostal muscles, the levatores costarum also act in raising the ribs and in increasing the thorax in the transverse and antero-posterior diameters.

These are the essential muscles of inspiration, but other muscles also participate in the act. In many individuals, even when breathing quietly, it will be seen that the nostrils dilate with each inspiration. This is due to the action of the dilatores narium, which contract synchronously with the other muscles of inspiration. Again, if the larynx be examined, it will be found that the vocal cords slightly diverge from one another during inspiration. This is brought about by the action of the posterior crico-arytenoid muscles (p. 50).

Forced Inspiration.—This comparatively small group of muscles is sufficient to carry out the ordinary act of inspiration. But, in certain conditions, inspiration becomes forced. A forced inspiration may be made voluntarily, often it is produced involuntarily. Such forced inspiration is well seen in patients suffering from heart disease, in whom the blood is not properly oxygenated, and by whom powerful efforts are made to get as much air into the lungs as possible. In this condition every muscle which can act upon the thorax to expand it is brought into play. The body and spinal column are fixed in the erect position. The head is thrown back and fixed by the posterior spinal muscles. The arms and shoulders are fixed—usually by holding on to the sides or arms of the chair—and every muscle which can act from the fixed spine, head and shoulder girdle upon the thorax is brought into play. Normally, these act from the thorax upon the parts into which they are inserted; now they act *from* their insertion *upon* their point of origin. The sterno-mastoids, sterno-thyroids, and sterno-hyoids assist in elevating the thorax. The serratus magnus, pectoralis minor, and upper fibres of the pectoralis major, and the part of the latissimus dorsi which passes from the humerus to the three last ribs, also pull these structures upwards. The facial and laryngeal movements also become exaggerated.

B. In *Expiration* the various muscles of inspiration cease to act, and the forces against which they contended again contract the thorax in its three diameters—

The *elasticity of the lungs* is no longer overcome by the muscles of inspiration, and the external atmospheric pressure acting along with it drives the chest wall inwards.

The *elasticity of the costal cartilages* and—in the erect position—the *weight of the chest wall* cause the ribs again to be depressed, and finally the *elasticity of the abdominal wall* drives the abdominal viscera against the relaxed diaphragm and again arches it towards the thorax, squeezing its marginal portion against the ribs and occluding the complemental pleura.

Experimental evidence shows that the internal intercostals contract with each expiration, and help to draw the ribs downwards.

Ordinary expiration is thus normally mainly a passive act, being simply a return of the thorax to the position of rest. But voluntarily, and, in certain conditions, involuntarily, expiration may be forced. Forced expiration is then partly due to the above factors, and partly due to the action of muscles. Every muscle which can in any way diminish the size of the thorax comes into play.

Chief of these are the abdominal muscles, which by compressing the viscera push them upwards and press the diaphragm further up into the thorax. At the same time, by acting from the pelvis to pull down the ribs, they decrease the thorax from side to side and from before backwards.

The serratus posticus inferior and part of the sacro-lumbalis pull downwards the lower ribs, and the triangularis sterni also assists in this.

By this constriction of the thorax, brought about by ordinary or by forced expiration, the air inside is compressed and the pressure raised. During ordinary expiration the highest pressure reached is about 2 to 3 mm. Hg, in forced expiration about 80 mm.

The pressure of the air outside is less than this, and the air inside the chest is driven out.

Special Respiratory Movements.—There are several peculiar and special reflex actions of the respiratory muscles, each caused by the stimulation of a special district, and each having a special purpose.

Coughing.—This consists of an inspiration followed by a strong expiratory effort during which the glottis is constricted but is forced open repeatedly by the current of expired air. It is generally due to irritation of the respiratory tract, and its object is to expel foreign matters.

Sneezing.—This is generally produced by irritation of the nasal mucous membrane, and its object is to expel irritating matter. It consists in an inspiratory act followed by a forced expiration during which, by contraction of the pillars of the fauces and descent of the soft palate, the air is forced through the nose.

Hiccough consists in a sudden reflex contraction of the diaphragm causing a sudden inspiration which is interrupted by a spasmodic contraction of the glottis. Abdominal irritation is its chief cause. It is allied to vomiting.

Sighing and *Yawning* are deep involuntary

inspirations which serve to accelerate the circulation of the blood when, from any cause, it becomes less active (*see* p. 38). They are probably due to cerebral anæmia, which they help to correct by increasing the general arterial pressure.

II. *Amount of Air respired.*—The amount of air respired is different in ordinary and in forced respiration.

In an ordinary respiration about 300 c.cms. of air enter and leave the chest. That is called the tidal air. Its amount varies with the size and muscular development of the chest.

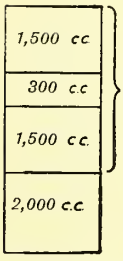


FIG. 146.—The amount of air respired in ordinary respiration, and in forced inspiration and expiration.

By a forced inspiration a much larger quantity of air may be made to pass into the lungs—a quantity varying with the size and strength of the individual—but on an average about 1500 c.cms. This is called the complemental air.

By forced expiration an amount of air much larger than the tidal can be expelled, an amount usually about the same as the complemental air, and called the reserve air.

The total amount of air which an individual can draw into and drive out of his lungs is a fair measure of the size and muscular development of the thorax, and it has been called the vital capacity of the thorax. This vital capacity may be measured by means of a *spirometer*. Its amount, as thus indicated, depends a good deal upon practice; the instrument, therefore, cannot be considered as of much practical value, and a more reliable conclusion as to the vital capacity may be arrived at by measuring the circumference of the chest in expiration and in inspiration.

Even after the whole of the reserve air has been driven out of the chest, a considerable quantity still remains in the air vesicles, its amount depending upon the size of the chest, but averaging about 2000 c.cms. This is called the residual air.

This very important point must always be remembered, that *the air taken into the chest never fills the air vesicles, and that air is never driven completely out of them. The air in them is thus not changed by the movements of respiration, but by the process of diffusion.*

III. *Interchange of Air in the Lungs by Diffusion of Gases.*—It has been shown that only the air in the trachea and bronchi undergoes exchange in mass, but that the air of the vesicles is not driven out of the chest. The renewal of this air depends upon the diffusion of gases.

If two gases are brought into relationship with one another they diffuse and tend to form a mixture uniform throughout. But if, at one point of a system, one gas is constantly being taken away and another constantly added, there will be a constant diffusion of the former *towards* the part where it is being taken up, and a constant diffusion of the latter *away from* the point to which it is being added. Suppose a tube (Fig. 147) containing oxygen and carbon dioxide, and suppose that at one end of the tube oxygen is constantly being taken up and carbon dioxide constantly given off, a diffusion of the gases in the direction indicated by the arrows will continually go on, and thus a constant supply of oxygen will be conveyed to the bottom of the tube, while carbonic acid will constantly be cleared out. This is exactly the condition in the lungs.

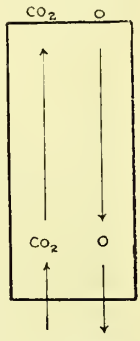


FIG. 147.—Shows the course of diffusion of oxygen into, and of carbon dioxide out of, the air vesicles.

The lower part of the tube corresponds to the air vesicles—the upper part to the air-passage in which the air is constantly being exchanged by the movement of respiration.

The mechanism by which the gaseous exchange in the alveoli is carried out is thus a double one.

IV. *Breath Sounds.*—The air as it passes into and out of the lungs produces sounds, which may be heard on listening over the thorax. The character of the breath sounds is of the utmost importance in the diagnosis of diseases of the lungs, and must be studied practically.

On listening over the trachea or over the bifurcation of the bronchi behind (between the 4th and 5th dorsal vertebræ), a harsh sound, something like the guttural *ch* (German *ich*), may be heard with inspiration and expiration. This is called the bronchial sound.

If the ear be applied over a spot under which a mass of air vesicles lies, a soft sound, somewhat resembling the sound of gentle wind among leaves, may be heard throughout inspiration, and for a third or less of expiration. This is called the vesicular sound.

When the air vesicles become consolidated by disease, the vesicular sound is lost and the bronchial sound takes its place. The cause of the vesicular character is therefore to be sought in the vesicles, infundibula, or small bronchi.

The cause of the bronchial sound has been determined by experiments on horses. In the study of the cardiac circulation it was shown that a column of fluid—and the same is true of a column of air—moving along a tube of uniform calibre, or with the calibre only slowly changing, produces no sound. Any sudden alteration in calibre produces vibration and a musical sound, as explained on p. 20. The first sharp constriction of the respiratory tract is at the glottis, and it is here that the bronchial sound is produced. If the trachea is cut below the larynx

and drawn freely outwards, the bronchial sound at once stops and the vesicular sound becomes lower and less distinct.

The cause of the vesicular sound is not so satisfactorily explained. It is in part due to propagation of the bronchial sound, altered by passing through vesicular tissue; but it is also probably due to the expansion and contraction of the air vesicles drawing in and expelling air, either through their somewhat narrow openings into the infundibula, or through the narrow opening of the infundibula into the bronchioles. The reason why the sound is best heard during inspiration may be that the sound is best conducted in the direction of the air stream (Fig. 142).

V. *Rhythm of Respiration.* — These movements of respiration are carried on in a regular rhythmic manner. Their rate varies with many factors; but the average number of respirations per minute in the adult male is about sixteen, or about one to every four or five beats of the heart. The rate of respirations may be modified by the will, and therefore, in counting the respirations in a patient, it is well to prevent his being aware of what is being done. Similarly, on account of this influence of the upper part of the brain, the respirations should not be counted while the patient is excited or nervous.

The most important factor modifying the rate of respiration is the age of the individual. The following table gives the average rate at different ages:—

Under 1 year . .	44
„ 5 years . .	26
„ 20 „ . .	19
Adult	16

The other modifications in the rate of breathing will be better understood after studying the nervous mechanism of respiration.

In these respiratory movements the phase of inspiration bears a certain proportion to that of expiration. Inspiration is much more rapid than expiration (*see* Fig. 149). As soon as inspiration is completed, a reverse movement occurs, which is at first rapid, but gradually becomes slower, and may be followed by a pause, during which the chest remains in the collapsed condition. The existence and duration of this pause varies much, and it may really be considered as the terminal period of expiration. Considering it in this light, we may say that inspiration is to expiration as 6 is to 7.

VI. *Controlling Mechanism of Respiration.*— The rhythmic movements of respiration require the harmonious action of a number of muscles, and this is directed by the nervous system.

The diaphragm is supplied by the phrenic nerves rising from the third and fourth, and partly from the fifth cervical nerves. The intercostals are supplied by branches from their corresponding dorsal nerves.

If the spinal cord be cut below the fifth cervical nerve the intercostal muscles cease to act. If the section is made above the third cervical nerve, the diaphragm, too, is paralysed, and the animal dies of suffocation.

Respiratory Centre.—Obviously, then, there is some nervous mechanism above the spinal cord presiding over these muscles.

Removal of the brain above the medulla oblongata does not stop the respiratory rhythm.

The mechanism must, therefore, be situated in the medulla oblongata.

If the medulla is split into two by an incision down the middle line, respiration continues, but the two sides do not always act at the same rate. The mechanism, then, is bilateral, but normally the two parts are connected, and thus act together.

Destruction of the part of the medulla lying near the root of the vagus arrests respiration, and it may therefore be concluded that the nervous mechanism presiding over this act is situated there.

It must not be imagined that this centre sends fibres directly to the muscles concerned in respiration. The nerves passing to these come from the cells in the grey matter of the spinal cord, and it is by influencing the activity of these cells that the respiratory centre controls the act of respiration.

Since expiration, when forced, is a complex muscular act, it is reasonable to suppose that the respiratory centre contains two parts—one presiding over inspiration, one presiding over expiration. While the inspiratory centre is constantly in rhythmic action, the expiratory centre is only occasionally at work.

Mode of Action of the Respiratory Centre.— Both parts of the respiratory centre are under the control of higher nerve centres, and through these they may be thrown into action at any time, or even prevented from acting for the space of a minute or so. But, after the lapse of this period, the respiratory mechanism proceeds to act in spite of the most powerful attempts to prevent it.

The activity of the centre is chiefly regulated by the amount of CO_2 in the blood going to it, and everything which leads to an increase in the CO_2 increases the activity of respiration. So perfect is this mechanism that the percentage of CO_2 in the air in the lungs is kept very constant in different conditions.

This is probably the explanation of a peculiar type of breathing, known as Cheyne-Stokes breathing, which sometimes occurs in heart disease and in other conditions, when the patient stops breathing for a time, then begins to breathe first quietly, then more forcibly, and after several respirations again with decreasing depth till the respirations stop. In these cases the respiratory centre is less excitable than usual, and is called into action only when CO_2

has accumulated in the blood. After this accumulation has been got rid of by the forcible respirations the activity of the centre again wanes.

Decrease in the amount of oxygen in the blood acts in the same way, but since the amount of oxygen in the arterial blood varies much less than the amount of CO_2, this factor plays a less important part in regulating breathing. At high altitudes the decrease in the pressure of oxygen in the air breathed, leading to its decreased amount in the blood, produces a marked increase of respiratory movements.

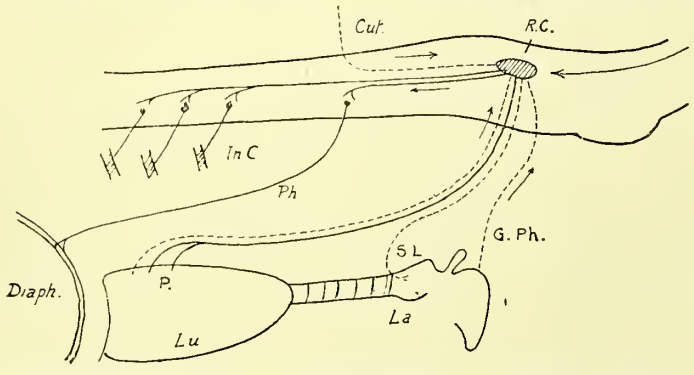


FIG. 148.—Nervous mechanism of respiration. *R.C.*, Respiratory centre; *Cut.*, cutaneous nerves; *Ph.*, phrenics; *In.C.*, intercostal nerves; *P.*, pulmonary branches of vagus; *S.L.*, superior laryngeal branch of vagus; *La.*, the larynx; *G.Ph.*, glossopharyngeal nerve; *Diaph.*, diaphragm.

But the respiratory centre is also acted upon by various ingoing nerves.

Vagus.—Since the vagus is the ingoing nerve of the respiratory tract, we should expect it to have an important influence on the centre (Fig. 148).

Section of one vagus causes the respiration to become slower and deeper; but after a time the effect wears off and the previous rate and depth of respiration is regained (Fig. 149).

Section of both vagi causes a very marked slowing and deepening of the respiration, which persists for some time, and passes off slowly and incompletely. But if, after the vagi have been cut, the connection of the centre with the *upper brain tracts* is severed, the mode of action of the centre changes. Instead of discharging rhythmically, it may discharge irregularly.

To investigate further this influence of the vagus it is necessary to study the effect of stimulating the nerve.

Strong stimulation of the pulmonary branches of one vagus (vagus below the origin of the superior laryngeal) causes the respiration to become more and more rapid, the inspiratory phase being chiefly accentuated. If the stimulus is very strong, respirations are stopped in the phase of inspiration. Weak stimuli, on the other hand, may cause inhibition of inspiration.

Such experiments prove that impulses are constantly travelling from the lungs to the centre, regulating the rhythmic activity of the centre.

How do these impulses originate in the lungs? Apparently from their alternate expansion and contraction.

If the lungs be forcibly inflated—*e.g.* with a bellows—the inspiration becomes feebler and feebler, and finally stops. The nature of the gas, if non-irritant, with which this inflation is carried out is of no consequence. If, on the other hand, the lungs be collapsed by sucking air out of them, the inspiration becomes more and more powerful, and may end in a spasm of the inspiratory muscles.

This shows that with each expiration a stimulus passes up the vagus which acts upon the inspiratory centre to make it discharge. The vagus is thus an excito-motor nerve, making the centre act in a reflex manner. With each collapse of the lung the vagus is thrown into action; as the lungs expand it ceases to act, and, as a result, the inspiratory centre stops acting, the muscles of inspiration cease to contract, and expiration occurs.

Action of other Ingoing Nerves on the Respiratory Centre.—The upper part of the respiratory tract, the larynx, receives its sensory fibres from the vagus. Section of the superior laryngeal branch of the vagus does not alter the rhythm of respiration. Stimulation of the upper end of

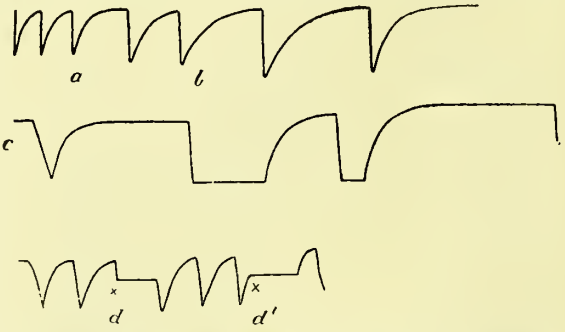


FIG. 149.—Tracings of the respirations—downstroke is inspiration; upstroke is expiration. At *a* one vagus nerve was cut; at *b* the second was divided; at *c* the upper brain tracts also were cut off; *d* and *d'* show the effect of stimulating the glossopharyngeal nerve.

the cut nerve causes first an inhibition of inspiration, and, if stronger, produces forced expiratory acts. This is well illustrated by the very common experience of the effect of a foreign body, such as a crumb, in the larynx. The fit of coughing is a series of expiratory acts produced through this nerve.

Another set of visceral nerves having an important influence on the respiratory rhythm are the splanchnics. When these are stimulated inspiration is inhibited. Everyone has

experienced the "loss of wind" as the result of a blow on the abdomen.

The glossopharyngeal, which supplies the back of the tongue, when stimulated, as by the passage of food in the act of swallowing, causes an instant arrest of the respiratory movements either in inspiration or expiration. The advantage of this in preventing the food as it is swallowed from passing into the trachea is obvious (Fig. 149, *d* and *d'*).

Stimulation of the Cutaneous Nerves stimulates the inspiratory centre and causes a deep inspiration. This is seen when cold water is dashed upon the skin, and is more clearly demonstrated in animals with the vagi and upper nerve tracts cut across. In such animals if the skin be touched an inspiratory movement is made.

The temperature of an animal also acts on the respiratory centre. Increase in temperature accelerates the rate of the heart—so, too, it accelerates the rate of the respirations, and in about the same proportion, as is seen in feverish attacks, where pulse and respiration are proportionately quickened so that their ratio remains unaltered. When the respiratory rate rises out of proportion to the rate of the pulse, it is usually an indication that some pulmonary irritation is present.

VII. *Interaction of Circulation and Respiration.*—The lungs and heart being packed tightly together in the air-tight thorax, and both undergoing periodic changes, necessarily influence one another. At the same time, the close proximity of the respiratory and cardio-motor centres in the medulla seems to lead to the activity of one influencing the other.

Influence of Respiration on Circulation.—The circulation is modified in two ways by respiration. First, the rate of the heart, and second, the arterial blood pressure undergo alterations.

1*st. Rate of Heart.*—If a sphygmographic trace giving the pulse waves during the course of two or three respirations be examined, it will be found that during inspiration the heart is acting more rapidly, while during expiration its action is slower.

If the vagus be cut these changes are not seen, showing that the inspiratory acceleration is not the result simply of the larger amount of blood which enters the heart during inspiration, but is really due to changes in the cardio-motor centre—the accelerating part of which has its activity increased during inspiration, while the inhibitory part is more active during expiration. This is, therefore, a reflex effect from the lung through the vagus, and it may be in part due to the proximity of the centres in the medulla.

But not only is the pulse more rapid during inspiration and slower during expiration, but the waves are smaller during inspiration and larger during expiration. The size of the wave depends much upon the pressure of blood in the arteries, and this change in the pulse thus leads to the fuller consideration of the changes in the arterial pressure due to respiration.

2*nd. Changes in Blood Pressure.*—If a tracing of the arterial pressure and of the respiratory movements be taken at the same time, it is found that there is a general rise of pressure during inspiration and a general fall during expiration, but that at the beginning of inspiration the pressure is still falling, and at the beginning of expiration it is still rising. This influence of respiration on arterial pressure is chiefly a mechanical one, depending on the variations in the pressure in the thorax during inspiration and expiration.

During inspiration the pressure in the thorax falls to below the atmospheric pressure, and thus during this period the heart and great vessels are under a diminished pressure. This diminution in pressure has little influence on the thick-walled ventricles and arteries, but tells markedly on the thin-walled auricles and veins. In these there occurs a diminution in pressure, which, in the case of the vena cava, may fall below an atmospheric pressure, and as a result an increased flow of blood into these vessels from the veins outside the thorax takes place (Fig. 141).

But when more blood enters the heart the activity of the organ is increased, and more blood is pumped through it into the arteries, and the pressure in these rises. This explains the great rise in arterial blood pressure during inspiration.

During expiration the pressure in the thorax rises to above the atmospheric pressure, and thus the pressure on the vessels in the thorax is increased. This tells on the thin-walled veins and auricles; and thus the flow of blood into them is retarded (Fig. 149), and less blood passing into the heart, less is pumped into the arteries, and the arterial pressure falls.

This, however, does not explain the slight fall of pressure at the beginning of inspiration, or the slight rise at the beginning of expiration. To understand these, the action of the pulmonary circulation has to be taken into account.

As inspiration develops the lungs are dilated, and the capillaries in them are also expanded. These expanding capillaries require more blood to fill them. They are situated on the course of the blood from the right side to the left side of the heart, and thus blood is retained from this stream to fill them, and less blood passes on into the left side of the heart, and out into the arteries, and a small fall in the arterial pressure occurs at the beginning of inspiration (Fig. 149).

Similarly, at the beginning of expiration the lungs are compressed and their blood-vessels squeezed, and thus the blood is driven out from them. Now, this blood cannot pass back into

the right side of the heart, so it must pass on into the left side. More blood is thus driven into the arteries, and the pressure rises. As soon, however, as the excess of blood has been squeezed out of the lungs, the contracted state of the vessels further retards the passage of blood to the left side of the heart and assists in diminishing the arterial pressure.

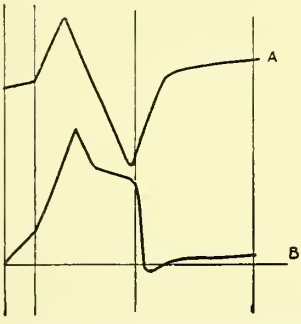


FIG. 150.—To show relations of cardio-pneumatic movements, *A*, to the cardiac cycle, *B*. In *A* the up-strokes are expiratory, the down-strokes inspiratory.

Influence of the Action of the Heart on Respiration.—The heart lies in the thorax surrounded by the elastic lungs. As it contracts and dilates it must alternately pull upon and compress the lungs, and thus tend to cause an inrush and an outrush of air—the cardio-pneumatic movements.

If a simultaneous tracing of the heart-beat and of the movements of the air column be taken, it will be seen that (1) at the beginning of ventricular systole there is a slight outrush of air from the lungs, probably caused by the blow given to the lungs by the suddenness of the systolic movement. (2) This is followed by a marked inrush of air corresponding to the outflow of blood from the ventricles, and caused by the fact that the contracting ventricles draw on and expand the lungs. (3) Succeeding this is a slower outrush of air corresponding to the active filling of the ventricles during the beginning of ventricular diastole. (4) Lastly, during the period of passive diastole, the cardio-pneumatic movements of air are in abeyance.

These cardio-pneumatic movements are of great importance in animals which hibernate. During their winter sleep the ordinary respirations almost stop, but a sufficient gaseous interchange is kept up by these cardio-pneumatic movements.

In the examination of the heart sounds they must be borne in mind, because, if there is any constriction in a small bronchus near the heart, the rush of air through it may give rise to a murmuring sound, in character very like a cardiac murmur and synchronous with the heart's action. On making the patient cough, such a murmur at once disappears.

B. *Interchange between the Air breathed and the Blood in the Lung Capillaries*

I. *Effect of Respiration upon the Air breathed.*—To determine this, some method of analysing the air exhaled must be employed. For this purpose the expired air is caught in a graduated vessel in which its amount can be measured; then a measured amount is forced into a chamber containing caustic potash, by which the CO_2 is absorbed, and the volume of air is again measured. It is now forced into a chamber containing pyrogallate of soda, which absorbs the oxygen and is again measured. The residue is nitrogen. In this way the amount of the gases present is determined.

The following table shows the average percentage composition of the air inspired and the air expired (Fig. 151)—

Per Cent of	N.	O.	CO_2.
Inspired air . .	79	21	0
Expired air . .	79	17	4

i.e. about 4 per cent of oxygen is taken from the air, and *about* 4 per cent of carbon dioxide is added to it. In man the amount of carbon dioxide given off is smaller than the amount of oxygen taken up. The proportion between the $\frac{CO_2 \text{ given off}}{O \text{ taken up}}$ is called the *Respiratory Quotient*, and it is thus less than unity—usually about ·8 to ·9—that is, for every five volumes of oxygen taken up only four volumes are given off in carbon dioxide, the remainder being combined with hydrogen to form water. The various factors modifying this quotient will be considered while dealing with the extent of the respiratory changes (*see* p. 49).

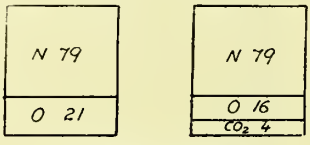


FIG. 151.—Shows the composition of inspired and expired air.

Expired air is saturated with watery vapour, and therefore it usually contains more *water* than inspired air.

Expired air also contains small amounts of *organic matter*, which may give it an offensive odour. These may possibly be to a small extent formed in the lungs, but they are to a greater extent produced by putrefactive change in the mouth and nose. It is probable that the accumulation of these products in the air is one of the causes of tbe injurious effect of the "foul air" in overcrowded spaces, but the evidence on this point is inconclusive.

Expired air is *usually* warmer than inspired air, because usually the body is warmer than the surrounding atmosphere. When, however, the temperature of the air is higher than that of the body, the expired air is cooler than the inspired.

This may be illustrated by the figures of an experiment—

Temperature of Inspired Air.	Temperature of Expired Air.
6·3° C.	30° C.
17–19° C.	37° C.
41° C.	38° C.
44° C.	38·5° C.

II. *Effect of Respiration on the Blood.*—To understand these changes in the air we must

refer to the changes in the gases of the blood in passing through the lungs (p. 6). Analyses show that the blood going to the lungs is poorer in oxygen and richer in carbon dioxide than the blood coming from the lungs (Fig. 152). *Oxygen is taken by the blood from the air, carbon dioxide is given by the blood to the air.*

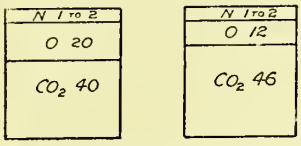


FIG. 152.—Shows the difference in the gases of arterial and venous blood.

How is this effected? The extensive capillary network in the walls of the air vesicles, if spread out in a continuous sheet, would present a surface of about 75 square metres. Between the blood in the capillaries and the air in the air vesicles are two layers of living cells—

1*st.* The endothelium lining the capillaries.

2*nd.* The flattened cells lining the air vesicles. Through these cells the interchange of gases must take place.

The interchange might take place in two different ways—

1*st.* By simple mechanical diffusion.

2*nd.* By some special action of the cells.

If the process follows strictly the laws of simple diffusion, it is then unnecessary to invoke the activity of the cells as playing a part. But if the gaseous interchange does not *strictly* follow these laws, we must conclude that the cells do play a part.

To determine if the process can be accounted for by diffusion, it is therefore necessary to know—

1*st.* The partial pressure or tension of the gases in the blood going to and coming from the lungs.

2*nd.* The partial pressure of the gases in the air in the air vesicles.

1*st. The Partial Pressure of the Gases in the Blood.*—Whether a gas is simply dissolved or whether it be held in loose chemical combination, the amount held will depend upon the *temperature* of the fluid and upon the *pressure* of the gas over the fluid. If the temperature is raised the fluid will hold less of the gas in solution, and any chemical combination will tend to split, as is seen when carbonate of lime is heated and the carbon dioxide is driven off.

If the pressure of any gas over a fluid be increased it will be taken up by the fluid, if it is decreased the gas will tend to come off from the fluid, as occurs when a bottle of soda-water is opened. But the same law applies to such chemical compounds as carbonate of lime. If it is heated in ordinary air—*i.e.* under a low pressure of carbon dioxide—the gas is given off; but if the lime is in an atmosphere of CO_2 the gas does not come off, but is taken up and the carbonate is formed.

It will thus be seen that for every temperature there is a certain pressure of the gas at which the solution or chemical combination will neither give off nor take up more of the gas. This may be determined by exposing the material in a series of chambers to air containing different proportions of the gas, and ascertaining by analyses of the air whether the gas has been given off or taken up, or has remained unaltered. The presence of a moist membrane between the fluid and the air makes no difference to these interchanges.

For blood, some form of aerotonometer is used, an instrument in which blood is allowed to trickle through a tube filled with air of known composition till the tensions of the gases in the blood and air have become the same. The tension is then determined by analysis of the proportions of the gases in the air in the tube.

We know that the pressure of gas in an atmosphere depends upon the proportion present. Suppose an atmosphere contains 20 per cent of oxygen, then the pressure, or partial pressure, of the oxygen is got by multiplying the percentage amount of the gas by 760—*i.e.* a whole atmosphere's pressure—and dividing by 100—

$$\text{Pressure of oxygen} = \frac{20 \times 760}{100} = 152 \text{ mm.}$$

The results obtained by means of the aerotonometer in the hands of different investigators have varied very widely.

Another method of arriving at the tension of oxygen in the blood has been devised by Haldane. It depends upon the fact that if hæmoglobin is exposed to a mixture of air with a trace of carbon monoxide in it, the amount of carbon monoxide taken up depends upon the oxygen tension — the greater the tension of oxygen the less CO will be taken up, and *vice versa*. Therefore if a person be allowed to breathe an atmosphere containing a known amount of CO, the amount of CO taken up by the blood will indicate the oxygen tension in the blood.

By such methods, although the results of different observers vary so greatly, it appears that in blood going to the lungs the O tension may be as low as 20 mm. Hg and the CO_2 tension as high as 40 mm. Hg; and that in blood coming from the lungs the O tension is generally well over 100 mm. Hg, while the CO_2 tension varies enormously, but is, on an average, about 20 mm. Hg.

2*nd. Partial Pressure of Gases in the Air Vesicle.*—The air in the alveoli is not renewed by direct ventilation from without, but by a process of diffusion (p. 42). For this reason the amount of oxygen in the alveoli must be much smaller, the amount of carbon dioxide much larger, than in the respired air.

Haldane has devised a method of procuring samples of the alveolar air for analysis. A wide tube is fitted with a measured glass bulb near

one end, and this bulb is made a vacuum. The end of the tube near the bulb is put in the mouth and the person under observation breathes through it. At the end of an ordinary inspiration he expires deeply and quickly through the tube, and by opening the upper

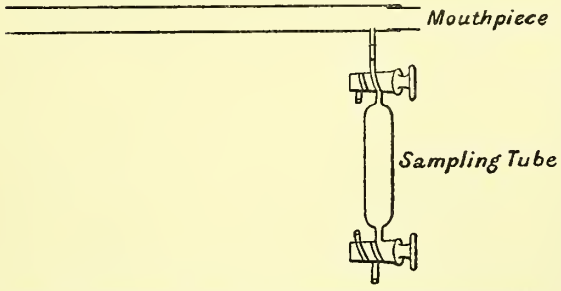


FIG. 153.—Haldane's apparatus for determining the composition of alveolar air.

stop-cock collects a sample of the expired air. A second sample is taken in the same way at the end of a normal expiration. The mean of these samples represents the average composition of the alveolar air.

By the use of this method it has been found that the partial pressure of the O varies within wide limits, while the partial pressure of the CO_2 remains very constant.

Thus at the top of Ben Nevis the pressure of oxygen was 76 mm. Hg; at the bottom of a mine it was 111; while in both places the pressure of carbon dioxide was about 42 mm.

In spite of these wide variations in the oxygen pressure in alveolar air the tension of oxygen in the blood remains about the same.

The difference in the pressure of these gases in the alveolar air, and in the blood going to and coming from the lungs, may be represented as follows in mm. Hg:—

	Oxygen		Carbon Dioxide	
Alveolar Air	76 to 111		42	
Blood	20 Venous	100+ Arterial	46 Venous	0–40 Arterial

This shows that when the blood reaches the lungs the distribution of pressure of the gases is such that, by the laws of diffusion, oxygen will pass from the alveolar air into the blood and carbon dioxide from blood to air; but that, before the blood has left the lungs, the distribution is such that oxygen *should*, by the laws of diffusion, pass *from blood to lungs* and carbon dioxide *from lungs to blood*, which is exactly the reverse of what occurs. The passage of oxygen to the blood and the passage of carbon dioxide from the blood is, in fact, much greater than could be accounted for by diffusion.

We must therefore conclude that the exchange of gases between the alveolar air and the blood *is not due entirely to diffusion, but is in part, at least, brought about by the activity of the cells lining the vessels and the alveoli.*

It should, however, be stated that, in spite of these figures, some physiologists maintain that simple diffusion will explain these interchanges—1*st*, because, when the amount of oxygen in the atmosphere—*i.e.* when the partial pressure of O—falls below a certain point, the gas is no longer taken up by the blood; and 2*nd*, because, when the amount and pressure of CO_2 rises, the CO_2 is not given off from the blood.

The partial pressure of oxygen may be reduced to a half without interfering with the vital processes of the body, and for this reason it is possible for men and animals to live at high altitudes. When men are suddenly subjected to a very marked decrease of pressure, especially if they have to do muscular work, as in climbing, the decreased supply of oxygen leads to shortness of breath, palpitation, and even to sickness (mountain sickness). These symptoms soon pass off, increased pulmonary ventilation and increased heart action increasing the intake of oxygen. Hence residence in high altitudes tends to increase the power of the respiratory muscles and the strength of the heart. It also increases the richness of the blood in erythrocytes and in hæmoglobin.

On the other hand, the atmospheric pressure may be enormously increased without any change in the metabolism. In a diving-bell 200 feet under water a pressure of 5120 mm. Hg—seven atmospheres—is sustained. As a result of the high pressure of the gases of the atmosphere they are dissolved in large quantities in the blood and tissues, and there is great danger in too sudden relief of pressure, since this may cause bubbles of gas to be given off in the vessels, and these may lead to air embolism and a plugging of the smaller vessels (Caisson disease).

B. Internal Respiration

1*st. The Passage of Oxygen from Blood to Tissues.*—In studying the physiology of muscle, which may be taken as a type of all the active tissues, it was seen that oxygen is constantly being built up into the muscle molecule, and that the living tissues have such an affinity for oxygen that they can split it off from such pigments as alizarin blue. The tension of oxygen in muscle is therefore always very low. We have seen that the tension of oxygen in arterial blood is nearly 100 mm. Hg; therefore, when the blood is exposed to a low tension of oxygen in the tissues, the oxygen comes off from the blood and passes into the tissues by the ordinary laws of diffusion.

But it must be remembered that this takes place in three stages:—

(1) The tissue elements are always taking up

oxygen from the lymph, because of the very low pressure of oxygen in the protoplasm, and because the protoplasm has an affinity for oxygen as for other nourishing substances.

(2) As a result of this, the oxygen pressure in the lymph falls and becomes lower than the oxygen pressure of the blood plasma, and thus the gas passes from the blood through the capillary walls to the lymph. How far this is simply the result of mechanical diffusion, and how far it is carried on by the vital action of the endothelium of the capillaries, we do not know.

(3) As a result of the withdrawal of oxygen from the plasma, the partial pressure round the erythrocytes is diminished, and the blood being at a high temperature, a dissociation of oxyhæmoglobin takes place, and the oxygen passes out into the plasma, leaving reduced hæmoglobin in the erythrocytes.

2nd. The Passage of Carbon Dioxide from Tissues to Blood.—The tissues are constantly producing carbon dioxide, so that it is at a high tension in them—about 60 mm. Hg. In the blood the carbon dioxide is combined with the sodium and is thus at a low tension. Hence there is a constant passage of carbon dioxide from the tissues to the blood.

C. Extent of Respiratory Interchange

The extent of the respiratory interchange in the lungs is governed by the extent of the internal respiratory changes—*i.e.* by the activity of the tissues. Every factor which increases the activity of the metabolic changes in the tissues increases the intake of oxygen and the output of carbon dioxide by the lungs.

It may be studied by enclosing an animal or a man in an air-tight case or chamber through which air of known composition is passed in measured quantity and analysed as it is withdrawn. This may be done by making the air pass through vessels containing sulphuric acid to absorb the water and caustic potash to fix the CO_2, and absorbing the oxygen by pyrogallate of soda.

In a normal man under average conditions the amount of exchange in twenty-four hours is about—

	Litres.	Grms.
Oxygen taken . .	540	770
Carbon dioxide excreted	432	850
Carbon ,,	...	230

That is, in one hour, on an average, he excretes 20 litres of CO_2 and absorbs 22 litres of O. The rate of exchange per unit of weight is more active in the child than in the adult.

1. *Muscular Work.*—Since muscle is the most abundant and active tissue of the body, muscular work more than anything else increases the respiratory changes (*see* vol. vii. p. 514).

2. *Food.*—The taking of food at once sets up active changes in the digestive apparatus. The muscular mechanism is set in action and the various glands secrete. As a result of these processes the respiratory interchange is at once increased. That the increase is dependent upon the increased functional activity of the digestive organs is shown by the fact that it is produced by the taking of substances which cannot be absorbed and used in the metabolic processes of the body.

But while the *immediate* increase in the respiratory interchange following the taking of food is due to the increased activity of the digestive structures, there is also an increase due to the utilisation in the body of the food taken. Whether proteins, fats, or carbohydrates, or all of these, form the diet, more oxygen is consumed and more carbon dioxide is given off than during starvation. The proportion between the oxygen taken and the carbon dioxide excreted is not, however, the same with all these food-stuffs. If the food is rich in carbon and poor in oxygen, a greater quantity of oxygen must be taken to oxidise it than if it is rich in oxygen.

The chief constituents of the food contain the following percentage amounts of oxygen and of carbon :—

	Oxygen.	Carbon.
Carbohydrates . .	53	40
Proteins . . .	22	52
Fats	12	76

Hence on a carbohydrate diet the respiratory quotient (p. 46) $\frac{CO_2}{O}$ is high, about 0·9 to 1, while on a fatty or protein diet it is low, 0·7 to 0·8.

3. *Temperature.*—If the temperature of the *body* be increased, the metabolic processes become more active and the respiratory interchanges are increased. But if the temperature of the *air* round the body be elevated, the metabolic processes may be diminished in activity, and the respiratory exchange decreased.

4. *Light.*—It has been shown that light increases the metabolic changes, and therefore the respiratory activity.

5. *Sleep.*—Since in sleep the animal is in a condition of muscular rest, since light is excluded from the eyes and since food is not taken, the respiratory exchanges are less active during sleep than during the waking hours. Similarly, in the long winter sleep of certain animals (hibernation), these factors, as well as the diminished temperature of the body, cause a great reduction in the intake of oxygen and output of carbon dioxide.

It is thus the internal which governs external respiration. Merely increasing the number or depth of the respirations has only a transient influence on the amount of the respiratory interchanges.

Ventilation.—The rate of gaseous exchange

governs the necessary supply of fresh air. It has been found that if the supply is insufficient, headache and sleepiness are apt to supervene, and experience has taught that each adult should have 2000 cubic feet of fresh air per hour. If 1000 cubic feet of space is allowed to each individual, the ordinary methods of exchange of air — through the chimneys and through chinks in windows and doors—should supply this quantity of air.

Asphyxia.—This is the condition caused by any interference with the supply of oxygen to the blood and tissues. It may be induced rapidly and in an acute form by preventing the entrance of air to the lungs, as in drowning or suffocation, or by causing the animal to breathe air deprived of oxygen, or by interfering with the flow of blood through the lungs, or with the oxygen-carrying capacity of the blood. It is slowly induced in a less acute form when the muscles of respiration fail as death approaches.

In *acute asphyxia* there is an initial stage of increased respiratory effort, the breathing becoming panting, and the expirations more and more forced. The pupils are small, and the heart beats more slowly and more forcibly, while the arterioles are strongly contracted, and a marked rise in the arterial pressure is produced. When the vagi are cut the slowing of the heart does not occur. Within a couple of minutes a general convulsion, involving chiefly the muscles of expiration, occurs. The intestinal muscles and the muscles of the bladder may be stimulated, and the fæces and urine may be passed involuntarily. Then the respirations stop, deep gasping inspirations occurring at longer and longer intervals. The pupils are dilated, and consciousness is abolished. The heart fails, and thus, although the arterioles are still contracted, the pressure in the arteries falls. Finally, the movements of the heart cease and death supervenes.

Voice

In connection with the respiratory mechanism of many animals, an arrangement for the production of sound or voice is developed. This is constructed on the principle of a wind instrument, and it consists of (1) a bellows, (2) a windpipe, (3) a vibrating reed, and (4) resonating chambers. In man and other mammals the bellows are formed by the lungs and the thorax. The trachea is the windpipe. The vocal cords in the larynx are the vibrating reeds, and the resonating chambers are the pharynx, nose, and mouth.

A. Structure of the Larynx.—The points of physiological importance about the structure of the larynx are the following:—

1. *Cartilages* (Figs. 154, 155).—The ring-like *cricoid* (*Cr.*) at the top of the trachea is thickened from below upwards at its posterior part and carries on its upper border two pyramidal cartilages triangular in section—the *arytenoids* (*Ar.*). These articulate with the cricoid by their inner angle. At the outer angle the posterior and lateral crico-arytenoid muscles are attached. From their anterior angles the vocal cords arise and run forward to the thyroid.

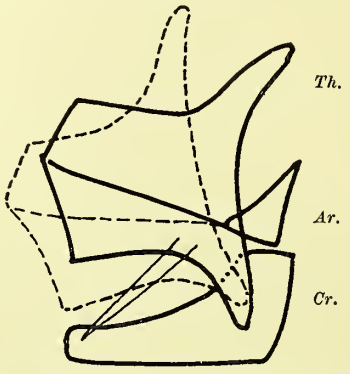


Fig. 154.—Side view of the cartilages of the larynx. *Cr.*, Cricoid cartilage; *Ar.*, right arytenoid cartilage; *Th.*, thyroid cartilage. The dotted line shows the change in the position of the thyroid by the action of the crico-thyroid muscle, and the stretching of the vocal cords which results.

The *thyroid* cartilage (*Th.*) forms a large shield which articulates by its posterior and inferior process with the sides of the cricoid, so that it moves round a horizontal axis. To the upper and anterior part, the *epiglottis* or cartilaginous lid of the larynx is fixed.

2. *Ligaments.*—The articular ligaments require no special attention. The true vocal cords are fibrous ligamentous structures which run from the arytenoids forward to the posterior aspect of the middle of the thyroid. They contain many elastic fibres and are covered by a stratified squamous epithelium and appear white and shining.

The vocal cords increase in length as the larynx grows, and in adult life they are generally longer in the male than in the female, and the whole larynx is larger.

3. *Muscles.*—The *crico-thyroids* take origin from the antero-lateral aspects of the cricoid, and are inserted into the inferior part of the lateral aspect of the thyroid. In contracting they approximate the two cartilages anteriorly, and render tense the vocal cords (Fig. 154).

The *crico-arytenoidei postici* arise from the back of the cricoid and pass outwards to be inserted into the external or muscular process of the arytenoids. In contracting they pull these processes inwards, and thus diverge the anterior processes and open the glottis (Fig. 155).

The *crico-arytenoidei laterales* take origin from the lateral aspects of the cricoid, and pass backwards to be inserted into the muscular processes of the arytenoids. They pull these forward and so swing inwards their anterior processes and approximate the vocal cords (Fig. 155).

A set of muscular fibres run between the arytenoids—the *arytenoidei*—while other fibres run from the arytenoids up to the side of the epiglottis. These help to close the upper orifice of the larynx.

The *thyro-arytenoid* is a band of muscular fibres lying in the vocal cords and running from the thyroid to the arytenoids. Its mode of action is not fully understood.

4. *Mucous Membrane.*—The mucous membrane of the larynx is raised on each side into a well-marked fold above each true vocal cord—

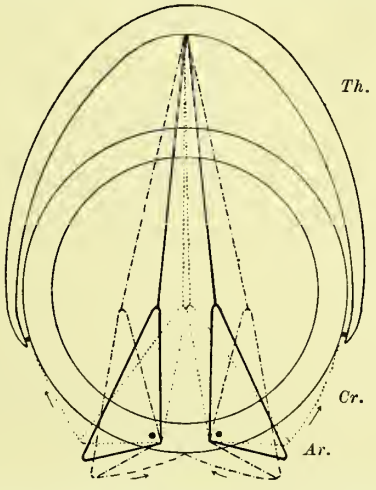


FIG. 155.—Cross section of the larynx, to show the cricoid, *Cr.*; thyroid, *Th.*; arytenoid cartilages, *Ar*. The continuous line shows the parts at rest, the dotted line under the action of the lateral crico-arytenoid muscle, and the dot-dash line under the action of the posterior crico-arytenoid.

the false vocal cord. Between this and the true cord on each side is a cavity—the ventricle of the larynx. The other folds of mucous membrane, although of importance in medicine, have no special physiological significance.

The interior of the larynx may be examined during life by the laryngoscope.

5. *Nerves.*—The muscles of the larynx are supplied chiefly by the recurrent laryngeal branch of the vagus which comes off in the thorax and arches upwards to the larynx. On the left side, where it curves round the aorta, it is apt to be pressed upon in aneurismal swellings. Paralysis of this nerve causes the vocal cord on that side to assume the cadaveric position, midway between adduction and abduction, and makes the voice hoarse or abolishes it altogether. The superior laryngeal is the great ingoing nerve, but it also supplies motor fibres to the crico-thyroid muscle. Paralysis prevents the stretching of the vocal cords, makes the voice hoarse, and renders it impossible to produce a high note.

Centre.—These nerves are presided over by (*a*) a centre in the medulla. When this is stimulated abduction of the vocal cords is brought about. (*b*) This centre is controlled by a cortical centre situated in the inferior frontal convolution. Stimulation of this causes adduction of the cords as in phonation, while destruction leads to no marked change.

B. PHYSIOLOGY. — When a blast of air is forced between the vocal cords they are set in vibration both wholly and in segments like other vibrating reeds, and sounds are thus produced. These sounds may be varied in loudness, pitch, and quality.

The loudness or amplitude of vibration depends upon the size of the larynx and upon the force of the blast of air acting on the cords.

The pitch, or number of vibrations per second, depends upon the length and tension of the vocal cords. The greater length of the vocal cords in the male, as compared with the female, makes the voice deeper. The tension of the cords may be varied by the action of the crico-thyroid muscle.

The power of varying the pitch of the voice differs greatly in different people. The average difference between the lowest and the highest note which the ordinary individual can produce is about two octaves.

The quality of the sound depends upon the overtones which are made prominent by resonance in the pharynx, nose, and mouth. By varying the shape and size of these cavities, and more especially of the mouth, the quality of sound may be considerably altered.

Singing Voice.—This is often classified by its average pitch and by its quality into bass and tenor in males, and contralto and soprano in females.

The true, or *chest, voice* is produced by a blast of air from the chest setting the cords into vibration, but the *falsetto voice*, which is generally higher in pitch than the true voice, is probably produced by vibrations of the edges of the cords.

Articulate Speech.—Spoken language is produced by varying the resonating chambers of the pharynx, nose, and mouth, and thus modifying the sounds produced in the larynx. *Whispered speech* is produced by setting in vibration in the resonating chambers a stream of air which has not been allowed to act upon the vocal cords.

In articulate speech two classes of sounds may be distinguished—(1) *Vowel Sounds.* These are musical sounds produced by a blast of air which is so modified by changing the shape of the mouth as to accentuate special overtones and thus produce the well-known *a*, *e*, *i*, *o*, *u* sounds (Fig. 156).

In sounding *a*, the mouth resembles a funnel with the wide part forward.

In *i*, it may be compared to a flask with the belly behind and the neck forward.

In *u*, the flask is reversed, the belly being forward.

In *o*, the mouth is intermediate between its position in *u* and *a*, and in *e* between *a* and *i*.

(2) *Consonant Sounds.*—These are more of the nature of noises—irregular vibration. They are produced either—(1) at the *lips*, (2) between the teeth and hard palate and the tongue, or (3) at the soft palate and back of the tongue. At these situations sounds may be produced—(1) by closing the orifice and then suddenly forcing it open, (2) by sending a current of air over a narrowing produced at one of these places, (3) by setting the edges of the narrowing in vibration.

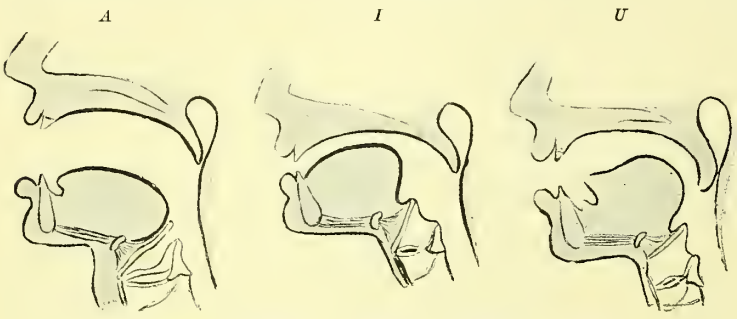


FIG. 156.—Changes in the shape of the mouth in sounding the vowels, *A*, *I*, *U*.

When the mouth is closed at one of these situations and air is forced through the nose, the nasal consonant sounds result.

Thus, according to their mode of production, the consonant sounds may be classified into explosives, aspirates, vibratories, or nasals.

II. Food and Digestion

I. Food.—The great use of food is to supply energy to the body. The muscles of the body are constantly active; they are constantly liberating energy by breaking down the complex molecules of proteins, fats, and carbohydrates, and hence a constant fresh supply of such material is necessary to prevent the body living on its own material and wasting away (*see* vol. vii. p. 514). During the growth of the body, too, the material from which it is to be built up, and the energy used in its construction, must be supplied by the food.

Hence a suitable food is one which will yield the necessary amount of energy, and will supply the materials necessary for repair and for growth.

But food must also supply the water and salts required to keep the various constituents of the body in solution, so that the essential chemical changes may go on.

Nature of Food

Food may be divided into foods not yielding energy and foods yielding energy.

A. *Food-Stuffs not yielding Energy.*—1. Water is the chief constituent. Since it is daily given off in large quantities by the kidneys, lungs, skin, and bowels, it must daily be supplied in sufficient amounts or the chemical changes cannot go on and death supervenes.

2. *Inorganic Salts.*—The most important of these is sodium chloride, which is essential for the maintenance of the chemical changes in the body. When it is not freely supplied in the food, it is retained in the tissues, and hence animals can, when necessary, live with a comparatively small supply.

3. *Salts of Organic Acids.*—The sodium and potassium salts of citric, malic, and tartaric acid, which are found abundantly in various vegetables, when taken into the tissues, are oxidised into carbonates which are strongly alkaline salts. The proteins which are decomposed in the body contain sulphur and phosphorus, and these are oxidised into sulphuric and phosphoric acids. In herbivorous animals the prejudicial effect of such acids is counteracted by the formation of these alkaline carbonates which neutralise the acids. In carnivorous animals these salts are not so necessary, since ammonium is formed from the nitrogen of the proteins in sufficient quantity to neutralise the acids produced. Man occupies a position midway between the herbivora and the carnivora. The amount of energy yielded by the breaking down of these salts into carbonates is so small that it is of no importance.

B. *Food-Stuffs yielding Energy.*—These are complex combinations of carbon, hydrogen, and oxygen, with or without nitrogen, sulphur, phosphorus, and iron. They are of the same nature as the materials which are found on analysis of dead protoplasm. They are commonly spoken of as the Proximate Principles of the food, and they may be classified as follows:—

1. Nitrogen-containing—Proteins and Sclero-proteins.

2. Non-nitrogenous—Fats and Carbohydrates.

In studying the value of these food-stuffs it is necessary to consider their Energy Value—that is, the amount of energy which can be yielded by the decomposition of a definite quantity of each in the body.

The fats and carbohydrates leave the body as carbon dioxide and water, the proteins leave it partly as carbon dioxide and water, partly as urea $CO(NH_2)_2$.

Such a body as glucose, $C_6H_{12}O_6$, by being oxidised to CO_2 and H_2O, gives off a certain amount of kinetic energy, and the amount of energy liberated is the same whether the oxidation is direct or takes place through any of many possible lines of chemical change.

In whatever ways a chemical substance breaks down into certain final products, the energy set free is always the same, and this principle is taken advantage of in determining the energy value of the food-stuffs. If fats and carbohydrates are changed to carbon dioxide and water

in the body, and if the energy given off as they undergo this change can be measured outside the body, their energy value as foods can be ascertained.

Determination of Energy Value.—This is done by burning a definite quantity of the material and finding how many degrees a definite quantity of water is heated. This gives the energy in *heat units*, and, by Joule's law, it can be converted into the equivalent for any other kind of energy, such as mechanical work.

It is known that the energy required to heat one kilogramme of water through one degree Centigrade is sufficient to raise 423 kilograms of matter to the height of one metre against the force of gravity, and thus, if the energy value of any material as a producer of heat is known in heat units—kilogram degrees or Calories—by multiplying by 423 we get the value in work units—kilogram metres (kgms.).

The apparatus used for ascertaining the heat produced by the combustion of material is called a calorimeter. Many different forms are in use, but the object in the water calorimeter is to secure that *all* the heat is used in raising the temperature of a known volume of water.

The value of the three great proximate principles of the food must be considered in detail.

1. *Proteins.*—The chemistry of these bodies has been already considered (vol. vii. p. 490). They are the "chief substances" of living matter, forming about 80 or 90 per cent of its dry residue. The molecule is one of great complexity, and contains C, H, O, N, S, and sometimes P. and Fe.

It is from the proteins of the food alone that the nitrogen and sulphur required in the construction and repair of the living tissues are obtained. The carbon and hydrogen required are also contained in these substances; and, as will be presently shown, they have a considerable energy value. Hence *Proteins form the essential organic constituents of the food.* Theoretically it should be perfectly possible for an animal to live on proteins alone, with a suitable addition of water and salts.

In estimating the actual energy value of proteins in the body a difficulty arises in the fact that, instead of being decomposed to CO_2, H_2O, NH_3, SO_3, as they are during combustion in the calorimeter, in the body the nitrogenous part is not broken down further than to urea—$CO(NH_2)_2$, 3 grms. of protein yielding 1 grm. of urea. If the energy value of the complete combustion of a definite amount of proteins is first ascertained, and then the energy value of the amount of urea derived from the same amount of protein determined, by subtracting the latter from the former, the energy value of proteins in their decomposition in the body is found (*see* p. 54).

The combustion of 1 grm. of protein to urea yields 4·1 Calories of Energy.

2. *Sclero-proteins.*—In studying the chemistry of the formed material of the various protecting, supporting, and connecting tissues, these substances have been considered (*see* vol. vii. p. 492).

Keratin, elastin, and mucin seem to be of no importance as articles of food. If taken in the food they pass through the alimentary canal practically unchanged.

While raw collagen seems also to be of little use, gelatin, formed by boiling collagen, has a certain value. Although it cannot take the place of proteins, because it cannot be used for building up the living tissues of the body, it is nevertheless decomposed into urea, and in decomposing it yields the same amount of energy as the proteins. It has, therefore, a definite though restricted value as a food-stuff.

3. *Fats.* — The chemistry of the fats has already been considered (vol. vii. p. 498). From the fact that they contain so little oxygen in proportion to their carbon and hydrogen, a large amount of energy is liberated in their combustion, and therefore they have a high energy value as food-stuffs.

One gramme of fat yields twice as much energy as the same amount of protein or carbohydrate. The combustion of 1 gramme of fat yields 9·3 Calories of Energy.

4. *Carbohydrates.* — The carbohydrates — starches and sugars—form a group of bodies which do not occur largely in animals, but are abundant constituents of plants.

They contain carbon, hydrogen, and oxygen, the carbon atoms of the molecule usually numbering six or some multiple of six, and the hydrogen and oxygen being in the same proportions in which they occur in water. They are aldoses or ketoses and derivations from these, of the hexatomic alcohol, $C_6H_{14}O_6$ (p. 93). A group of carbohydrates having five carbon atoms, and hence called Pentoses, have been found in the animal body, but they are of minor importance.

The simplest carbohydrates are the monosaccharids, of which dextrose, the aldose of mannite, is the most important. Dextrose is *the* sugar of the animal body. It has been called glucose, grape sugar, and blood sugar.

Closely allied to dextrose in chemical composition is the ketose *lævulose*, a sugar which, instead of rotating the plane of polarised light to the right, rotates it to the left, but which in other respects behaves like dextrose. It occurs in certain plants, and in the fœtus and fœtal fluids of certain animals.

The other monosaccharid of importance is *galactose*, a sugar produced by the splitting of milk sugar.

These monosaccharids, when boiled with a solution of cupric acetate in acetic acid (Barfoed's solution), are oxidised, taking oxygen from the cupric salt and reducing it to the cuprous state. When boiled with caustic potash, they, along

with certain of the double sugars, are oxidised, and if a metallic salt be present which can readily give up its oxygen, it becomes reduced, the sugar appropriating the oxygen. On this depends Fehling's and many other tests for glucose.

Under the influence of yeast they split into ethyl alcohol and carbon dioxide.

They also form crystalline compounds, osazones, with phenyl-hydrazin. These have proved most useful in distinguishing different sugars.

By the polymerisation of two monosaccharid molecules with the loss of water, disaccharids, or double sugars, are formed. Thus, two glucose molecules polymerise to form one *maltose* molecule.

$$C_6H_{12}O_6 + C_6H_{12}O_6 = 2(C_6H_{12}O_6) - H_2O$$
$$= C_{12}H_{22}O_{11}$$

Maltose is the sugar formed by the action of malt and other vegetable and animal zymins upon starch. By the action of dilute acids and other agents it can be split into two dextrose molecules. Like the monosaccharids, it ferments with yeast.

Lactose, the sugar of milk, is a disaccharid composed of a molecule of dextrose united to a molecule of galactose with dehydration. It readily splits into these two monosaccharids, but does not ferment with yeast.

Dextrose, polymerising with lævulose, yields *cane sugar*, and this sugar, so largely used as an article of food, can be split into dextrose and lævulose. It does not reduce Fehling's solution, and does not ferment with yeast. Under the action of mineral acids it breaks down into its component sugars.

By further polymerisation of monosaccharids with the loss of water, molecules of greater size are produced and form the set of substances known as the polysaccharids. Among the simplest of these are the *dextrins*.

Closely allied to dextrins are the *inulins*. But while dextrin is formed of dextrose molecules, inulin contains lævulose molecules. Both are formed from the splitting of the more complex *starches*. The molecule of soluble starch is built up of no less than thirty dehydrated monosaccharid molecules, and has a molecular weight of 9000. Ordinary starch seems to have a molecular weight of 20,000 or 30,000, and hence must be of still greater complexity.

These polysaccharids are distinguished from the sugars by being precipitated from their solutions by the addition of alcohol. They are not oxidised when boiled with caustic potash, nor do they change to alcohol and carbon dioxide under the influence of yeast. In cold neutral or acid solutions most of them strike a blue or brown colour with iodine. By boiling with a mineral acid and by the influence of various ferments they break down, take up water, and become monosaccharids—the starches yielding the dextroses, and inulins yielding lævulose.

Glycogen is animal starch. It gives an opalescent solution and strikes a brown colour with iodine.

The energy value of the carbohydrates is about the same as that of the proteins.

Energy Value of the Proximate Principles of the Food.—1 gramme of—

Protein yields . . .	4·1	Calories.
Carbohydrate yields .	4·1	,,
Fat yields . . .	9·3	,,

The Sources of the Various Proximate Principles of the Food

The proximate principles are in part derived from the *animal*, in part from the *vegetable* kingdoms. While some races procure their food entirely, or almost entirely, from the former, others depend almost entirely on the latter. The vast majority of mankind, however, use a mixture of animal and vegetable foods.

Animal foods may be classified as—

1. Milk and its products, Cream, Butter, and Cheese.
2. Flesh.
3. Eggs.

1. *Milk.*—The white colour of milk is due to its being a fine emulsion of fat globules floating in a clear plasma. Hence, when the cream is removed, milk becomes less white and less opaque. Its specific gravity is about 1030, and in man and herbivorous animals its reaction is alkaline.

The chief *protein* of milk is *caseinogen*, a nucleo-protein with a very small amount of phosphorus, which exists as a soluble calcic compound. It is held in solution in milk, but under the influence of various agents it clots or curds. Its calcic compound is split by the action of acids, and casein is precipitated. Under the influence of rennet caseinogen splits into whey albumin which remains in solution, and calcic paracasein which is insoluble and separates as a curd containing the fat. In cow's milk a small amount of an albumin is also present.

The *fats* of milk occur as small globules of varying size floating in the fluid, each surrounded by a protein envelope which must be removed by means of an alkali or an acid before the fat can be extracted with ether. The fats are chiefly olein, with smaller quantities of palmatin and stearin, and still smaller amounts of such lower fats as butyrin, capronin, and caprylin.

The *carbohydrate* of milk is lactose, a disaccharid, which splits into dextrose and galactose. Under the action of various micro-organisms it is split to form lactic acid, thus causing the souring of milk.

Phosphorous Compounds.—In addition to caseinogen, milk contains other organic phosphorous

compounds. Among these are *lecithin* and a compound which has been called *phosphocarnic acid*, the constitution of which is not fully understood. In human milk the greater part of the phosphorus is in organic combinations, while in cow's milk the amount in inorganic compounds is much greater.

Milk is especially rich in calcium and potassium, but the amount of iron in milk is very small, and therefore, when the child has used up the store of iron which it has in its body at birth, it is necessary to replace the milk-diet by foods containing more iron.

Cow's milk is an important constituent of the diet. Its *average* composition compared with human milk is as follows :—

	Cow's.	Human.
Water	88·3	88·8
Proteins	3·0	1·0
Fats	3·5	3·5
Carbohydrates . .	4·5	6·5
Salts	0·7	0·2

Butter and cream are simply the fats of the milk more or less completely separated from the other constituents.

Cheese is produced by causing the coagulation of the casein, which carries with it a large amount of the fats. If cheese is made before the removal of the cream it is rich in fats, if after the removal of the cream it is poor in fats. Cheese contains between 25 and 30 per cent of protein, and between 10 and 30 per cent of fat. It is as a source of protein that it is of chief value.

Cheese, when allowed to stand, affords a suitable nidus for the growth of micro-organisms by the action of which the proteins are digested into peptones and simpler bodies, and the fats split up into glycerine and the lower fatty acids. These free fatty acids give the peculiar flavour to ripe cheese. The lactose is in part converted into lactic acid.

2. *Flesh.*—Under this head may be included not only the muscles of various animals, but also such cellular organs as the liver and kidneys.

When free of fat, they contain about 20 per cent of proteins. These are chiefly native proteins, but a certain amount of collagen is also present which yields gelatin on boiling. The amount of fat may vary from almost *nil* in white fish to about 80 per cent in fat bacon. In animals specially fed, the amount of fat may be enormously increased, and even ordinary butchers' meat may have more fat than protein.

Flesh is thus a source of proteins and scleroproteins, and to a smaller extent of fats. The extractives include such bodies as creatin, xanthin, inosit, etc. (vol. vii. p. 503), which may help to give the peculiar flavour to the flesh of various animals.

Flesh may be preserved in various ways—*e.g.* by simply drying, by salting, or by smoking. The result of each of these procedures is to diminish the amount of water, and thus to increase the solids.

3. *Eggs.*—The egg of the domestic fowl need alone be considered. The composition of the white and of the yolk naturally differs considerably. The white of egg is nothing more than a solution of proteins.

In the yolk there is a very large amount of lecithin (p. 75) along with ordinary fats, and a large amount of a phospho-protein ; and the great value of eggs is thus that they contain both proteins, ordinary fats, and the special phosphorus containing protein and fat. The mixed contents of the egg contains a little more than 10 per cent each of proteins and of fats.

Speaking generally, we may say that the animal food-stuffs are rich in proteins and fats, but are poor in carbohydrates.

Vegetable Food-Stuffs. — In the food of man, vegetables play as important a part as animal products.

The peculiarity of special importance in vegetables is the existence of a capsule to the cells, composed of cellulose — a substance allied to starch in its composition, but which is very resistant to the action of the human digestive juices, and thus hinders the utilisation of the cell contents. In order that these may be digested and absorbed from the stomach and intestine, this capsule must be broken down either by some preparatory treatment, or by the teeth in the act of chewing. Although of practically no value as a food-stuff, it acts as a natural purgative by stimulating the intestines, and is of great value in keeping up the regular action of the bowels.

I. *Cereals.*—From the seeds of these, meals and flours are prepared.

Oatmeal contains about 15 per cent of proteins, about 6 per cent of fats, and about 65 per cent of carbohydrates.

Wheaten flour contains about 10 per cent of proteins, 1 per cent of fats, and 75 per cent of carbohydrates.

Ordinary *white bread*, prepared from wheaten flour, contains only about 7 per cent of proteins and 55 per cent of carbohydrates.

The proteins of these cereals are mixtures of various globulins which differ, in certain of their characters, from the animal proteins. They are most abundant in the outer part of the grain, just under the capsule.

While oatmeal and maize contain a fair proportion of fat, the other cereals are poor in this constituent.

The chief constituents of all these seeds are the carbohydrates stored as starch.

II. *Legumens.*—The seeds of the leguminosæ

—peas, beans, and lentils—are valuable constituents of the diet, being specially rich in proteins. In the dry state they contain something over 20 per cent of proteins and about 50 per cent of carbohydrates. The Soy bean of Japan contains no less than 34 per cent of proteins and 17 per cent of fats, with 34 per cent of carbohydrates.

The proteins are a mixture of albumins and globulins which have been classified together as legumin.

The fats are generally small in amount, and the carbohydrates, though abundant, are less so than in the cereals.

III. *Bulbous Plants.*—The underground stems of certain plants develop tuberous growths in which material for the nourishment of the plant is stored. These plants belong to different natural orders, but they may here be classified together. The most commonly employed are potatoes, turnips, and carrots. The amount of proteins is small, something under 2 per cent, while the carbohydrates in the potato reach 20 per cent, but in other tubers only about 10 per cent.

Such tubers are chiefly valuable as a source of carbohydrates—though they also contain a small proportion of proteins.

IV. *Green Vegetables.*—Cabbage, cauliflower, spinach, lettuce, etc., are useful additions to the diet, but their value as a source of the proximate principle of the food is not great on account of the amount of water they contain.

They are rich in the potash salts of the organic acids, the importance of which has already been discussed (p. 52). The cellulose forming the walls of the cells in the young and growing part of the leaves seems to be capable of partial digestion in the human intestine.

V. *Fungi.*—Mushrooms and other such fungi do not constitute a sufficiently important part of the diet to require attention.

VI. *Fruits.* — These vary considerably in composition. Most are rich in water and carbohydrates, poor in proteins, and contain practically no fat. Their great value lies in the amount of free and combined organic acids they contain. Bananas, which contain about 23 per cent of soluble carbohydrates, may be considered as a food of some importance, while dried fruits, especially dates and figs, which contain between 4 and 5 per cent of proteins and about 65 per cent of carbohydrates, are of considerable nutritive value.

VII. *Nuts.*—These, unlike the fruits, are for the most part poor in water and carbohydrates, but rich in proteins and fats. Chestnuts, however, contain an abundance of carbohydrates, and a smaller proportion of proteins and fats.

Cooking.—Few of these food-stuffs are used by civilised man in a raw and unprepared condition. With the object of rendering them more palatable and more easily digested, and also in order to destroy bacteria, they are usually cooked. This process of cooking produces important changes in many of the foods, and its effects must be briefly considered.

Milk and its products are practically unaltered by cooking.

Flesh is cooked either by exposing it directly to heat or by treating it with boiling water.

Roasting, grilling, broiling, and frying are modifications of the former method, and in all of them the heat at once coagulates the proteins at the outer part of the piece of flesh, and thus forms a more or less impermeable covering, which prevents the escape of the juices of the meat. Hence these methods of cooking, although bringing about a burning of the outer layer of the flesh, leave all the constituents in only slightly altered proportions.

On the other hand, if a piece of flesh be put into cold water and boiled, the proteins, the salts, and the various extractive bodies which give it its flavour are extracted, and, as the water warms, the fats also are dissolved out, and the meat becomes poorer in these constituents, while the surrounding water becomes a soup. In this soup the dissolved proteins precipitate as the temperature rises, and when the soup is cooled they rise to the top with the fats, and are generally removed as a scum. Hence soups are poor in the proximate principles of food, but rich in the extractives and salts of meat.

If, however, the piece of meat to be cooked is plunged into a large quantity of boiling water, the proteins at its outer part at once coagulate and form a covering which prevents the loss of the nutrient material which occurs when the meat is slowly boiled.

Stewing is a modification of boiling by which much of the nutrient material of meat is extracted, but this is served as a gravy.

It is in vegetables, however, that cooking is of the greatest importance, since by it the cellulose envelopes which enclose the digestible portions of the plant are ruptured.

II. Digestion

I. Structure of Alimentary Canal.—The anatomy and histology of the alimentary tract must be studied practically. A mere outline of the various structures such as will assist in the comprehension of their physiology is given here. The Alimentary Canal (Fig. 157) may be divided into the mouth, the œsophagus or gullet, the stomach, the small and large intestines, and three sets of supplementary structures—the salivary glands, the liver, and the pancreas.

The mouth, provided with its teeth, and surrounded by its mobile muscular wall, with the muscular tongue lying in its floor, is the part of the canal in which the food is broken up and prepared for digestion. Into the mouth

three pairs of compound glands—the salivary glands—open. The parotid, lined entirely by enzyme-secreting epithelium, opens on the side of the cheek, while the submaxillary gland, composed partly of acini with enzyme-secreting, and partly of acini with mucin-secreting epithelium, and the sublingual, composed entirely of mucin-secreting acini, open under the tongue (*S.G.*).

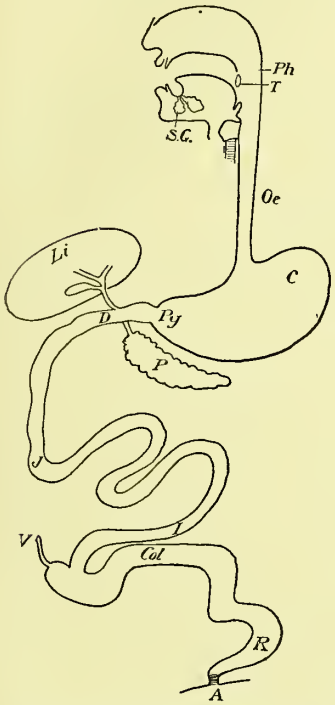


FIG. 157.—Diagram of the parts of the alimentary canal, from mouth to anus. *T.*, Tonsils; *Ph.*, pharynx; *S.G.*, salivary glands; *Oe.*, œsophagus; *C*, cardiac; *Py.*, pyloric portion of stomach; *D*, duodenum; *Li.*, liver; *P.*, pancreas; *J.*, jejunum; *I.*, ileum; *V.*, vermiform appendix; *Col.*, colon; *R.*, rectum.

The tongue is covered with a fine fur of processes, the *filiform papillæ*, which are of use in passing the food backwards along its surface in the act of swallowing. (For Organs of Taste, *see* vol. vii. p. 531.)

Posteriorly, the mouth opens into the pharynx (*Ph.*) or upper part of the gullet. On each side, between the mouth and the pharynx, is the tonsil (*T.*), an almond-like mass of lymphoid tissue. The pharynx is a cavity which can be shut off above from the posterior nares by raising the soft palate, and by pulling forward the posterior pharyngeal wall. It is surrounded by three constrictor muscles, which, by contracting from above downwards, force the food down the gullet, towards the stomach.

The œsophagus (*Oe.*) is a muscular-walled tube lined by a stratified squamous epithelium. The muscles, below the lowest constrictor of the pharynx, are of the visceral type, and are arranged in two layers, an outer longitudinal layer and an inner circular layer.

The stomach is a dilatation of the alimentary canal into which the gullet opens. To the left it expands into a sac-like cardiac end (*C.*), and to the right it narrows, forming the pyloric end (*Py.*). Like the gullet, it is surrounded by visceral muscular fibres, arranged essentially in two sets. At the cardiac orifice, the circular fibres form a not very marked cardiac sphincter, and at the pyloric end they form a very thick and strong pyloric sphincter.

The mucous membrane, which is covered by a columnar epithelium, is largely composed of tubular glands, those at the cardiac end containing two kinds of cells, the peptic and the oxyntic cells, those at the pyloric end containing peptic cells alone.

The Small Intestine is a tube of about 7 metres in length. It has a double muscular coat like the stomach. The mucous membrane, which is covered by a columnar epithelium, is thickly set with simple test-tube-like glands—*Lieberkühn's follicles*—and is projected into the lumen of the tube, as a series of delicate finger-like processes, the *villi*. The tissue of the villi and that between the Lieberkühn's follicles is chiefly lymphoid, and in certain places this lymphoid tissue is massed in nodules which are either placed singly or grouped together in the lower part of the small intestine to form Peyer's patches. In the first part of the small intestine—the upper part of the duodenum (*D.*)—the submucous layer is full of small branching glands lined by an enzyme-secreting epithelium (*Brunner's glands*).

The Large Intestine is about 2 metres in length. The small intestine enters it at one side, and the opening is guarded by a muscular sphincter and by a fold of mucous membrane which forms the ileo-cæcal valve. Above the opening of the small intestine a cæcal pouch exists, and at the top of this is the vermiform appendix (*V.*), a narrow tube with an abundance of lymph tissue in its wall. Below the opening of the small intestine is the colon (*Col.*), which, after passing up the right side across the abdomen and down the left side, takes an S-like bend to end in the rectum (*R.*), which, passing forward, suddenly turns down and opens at the anus. The sudden bend is of importance in retaining the contents of the rectum. The last part of the rectum is surrounded by a strong band of muscle—the internal sphincter ani—by which it is compressed. The whole large intestine is covered by columnar epithelium, and is studded with Lieberkühn's follicles, in which the epithelium is chiefly mucus-secreting in type. There are no villi. The muscular coat of the colon differs from that of the rest of the alimentary canal in that the longitudinal fibres are arranged in three bands.

Into the duodenum, the bile duct and the duct of the pancreas open. The bile duct is formed by the union of the ducts from the lobes of the liver. Upon its course is a diverticulum, the gall bladder. The Liver (*Li.*) is a large solid organ, formed originally as a double outgrowth from the alimentary canal. Each of these outgrowths branches repeatedly, and the blood coming from the mother to the fœtus flows in a number of capillary channels between the branches. Later, when the alimentary canal has developed, the blood from it is streamed between the liver tubules. In man and in other

mammals the fibrous tissue supporting the liver cuts it up into a number of small divisions, the lobules, each lobule being composed of a series of obliterated tubules arranged radially with blood-vessels coursing between them.

The portal vein which takes blood from the stomach, intestine, pancreas, and spleen breaks up in the liver (Fig. 115, p. 10), and carries the blood between the lobules. From the interlobular branches, capillaries run inwards and enter a central vein which carries the blood from each lobule, and pours it into the hepatic veins which join the inferior vena cava. The supporting tissue of the liver is supplied by the hepatic artery, and its terminal branches have a very free communication with those of the portal vein.

The Pancreas is essentially the same in structure as the parotid gland. But in the lobules are certain little masses of epithelium-like cells closely packed together, the Islets of Langerhans (Fig. 158).

The Nerve Supply of the Alimentary Canal.—The muscles round the mouth are supplied



FIG. 158.—Section of pancreas to show acini of secreting cells; a large duct; and in the centre an island of Langerhans.

by the fifth, seventh, and twelfth cranial nerves. The nerve supply of the salivary glands will be considered later. The pharyngeal muscles are supplied by the ninth and tenth cranial nerves, and the œsophagus is supplied by the tenth, and by fibres from the sympathetic.

The stomach and intestine get their nerve fibres from two sources (Fig. 138, p. 33), above the descending colon from the vagus and the abdominal sympathetic, and below this from the nervi erigentes and abdominal sympathetic—the various fibres passing through the abdominal sympathetic ganglia. In the wall of the stomach and intestine these nerves end by forming an interlacing set of fibres, with nerve cells upon them, from which fibres pass to the muscles and glands. One of these plexuses (Auerbach's) is placed between the muscular coats—the other (Meissner's) is placed in the submucosa. These plexuses resemble the nervous system of the lower invertebrates rather than the synaptic system of the central nervous mechanism.

II. PHYSIOLOGY

i. DIGESTION IN THE MOUTH.—A. *Mastication.*—In the mouth, by the act of chewing, the food is thoroughly broken up and mixed with saliva.

The muscular mechanism of mastication may be here briefly indicated.

Movements of Mastication.—Movements of Lower Jaw in—1. Vertical Plane. *a. Elevation.* α. Temporal. β. Masseter. γ. Internal Pterygoid. *b. Depression.* α. Weight of Jaw. β. Anterior Belly of Digastric. γ. Mylo- and Genio-Hyoid. δ. Platysma. *Hyoid fixed by*—Omo- and Sterno-hyoid, Sterno-thyroid, and Thyro-hyoid. 2. Horizontal Plane—*Forwards:* External Pterygoids. *Backwards:* Posterior Fibres of Temporal. *To Right:* Left External Pterygoid. Right Temporal (Posterior Fibres). 3. Other Muscles of Mastication—Buccinator and Orbicularis oris.

B. *Saliva.*—The saliva is formed by the salivary glands—the parotid, submaxillary, sublingual, and various small glands in the mucous membrane of the mouth.

Characters.—It is a somewhat turbid fluid which, when allowed to stand, throws down a white deposit consisting of shed epithelial scales from the mouth, leucocytes, amorphous calcic and magnesic phosphates, and generally numerous bacteria. Its specific gravity is low—generally about 1003. In reaction it is neutral or faintly alkaline.

Chemically it is found to contain a very small proportion of solids. The saliva from the parotid gland contains only about 0·4 per cent, while that from the sublingual may contain from 2 to 3 per cent. The sublingual and submaxillary saliva in man is viscous, from the presence of mucin formed in these glands, while the parotid saliva is free from mucin. In addition to mucin, traces of proteins are present, and with these proteins is associated the active constituent or enzyme of the saliva—*ptyalin.*

Saliva generally contains traces of potassium sulphocyanide, and in some pathological conditions this may be markedly increased.

The functions of the saliva are twofold:—

1. *Mechanical*, to moisten the mouth and gullet, and thus to assist in speaking, chewing, and swallowing Since the salivary glands are absent from aquatic mammals, and since in carnivorous animals saliva has no chemical action, it would appear that this is the important function.

2. *Chemical.*—Under the action of the ptyalin of the saliva, polysaccharids, like the starches,

are broken down into sugars. Like other enzyme actions the process requires the presence of water and a suitable temperature, and it is stopped by the presence of strong acids or alkalies, by various chemical substances, and by a temperature of over 60° C., while it is temporarily inhibited by reducing the temperature to near the freezing point. During the short time the saliva acts on the food in the mouth the conversion is by no means complete. The starch is first changed into the dextrins, giving a brown colour with iodine, and hence called *erythrodextrins*, then into dextrins which give no colour with iodine, *achroodextrins*, and lastly into the disaccharid *maltose* (*see* p. 54).

Physiology of Salivary Secretion.—In order to study the physiology of salivary secretion, a cannula may be inserted into the duct of any

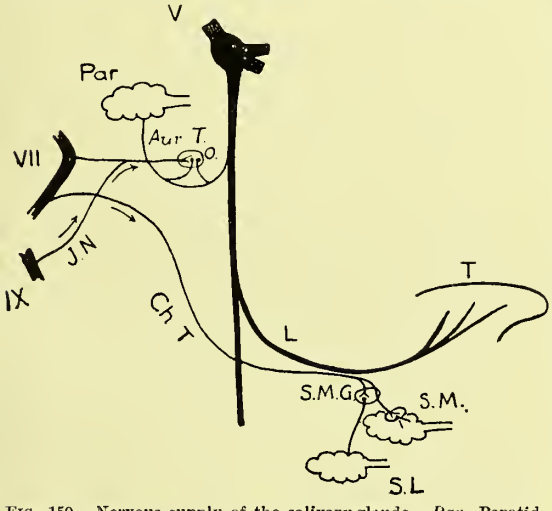


FIG. 159.—Nervous supply of the salivary glands. *Par.*, Parotid, and *S.M.* and *S.L.*, the submaxillary and sublingual glands; *VII.*, the seventh cranial nerve, with *Ch.T.*, the chorda tympani nerve, passing to *L.*, the lingual branch of *V.*, the fifth nerve, to supply the glands below the tongue, *T.*; *IX.*, the glossopharnygeal, giving off *J.N.*, Jacobson's nerve, to the *O.*, otic ganglion, to supply the parotid gland through *Aur.T.*, the auriculo-temporal nerve.

of the salivary glands and the rate of flow of saliva or the pressure of secretion may be thus measured. In this way it may be shown that the taking of food, or simply the act of chewing, and in some cases the mere sight of food, causes a flow of saliva. This shows that the process of secretion is presided over by the central nervous system.

The submaxillary and sublingual glands are supplied—(1) by branches from the lingual division of the fifth cranial nerve; and (2) by branches of the perivascular sympathetic fibres coming from the superior cervical ganglion. The parotid gland is supplied by the auriculo-temporal division of the fifth and also by sympathetic fibres (Fig. 159).

The influence of these nerves has been chiefly studied on the submaxillary and sublingual glands.

It has been found that when the lingual nerve is cut the reflex secretion of saliva still takes place, but that when the chorda tympani (*Ch.T.*), a branch from the seventh nerve which joins the lingual, is cut, the reflex secretion does not occur. Stimulation of the chorda tympani causes a copious flow of watery saliva, and a dilatation of the blood-vessels of the glands. If atropine has been first administered, the dilatation of the vessels occurs without the flow of saliva. This indicates that the two processes are independent of one another. The secreting fibres all undergo interruption before the glands are reached; the fibres to the sublingual gland having their cell station in the submaxillary ganglion (*S.M.G.*), the fibres to the submaxillary gland having theirs in a little ganglion at the hilus of the gland (*S.M.*). This was demonstrated by painting the two ganglia with nicotine (vol. vii. p. 550). When applied to the submaxillary ganglion the drug does not interfere with the passage of impulses to the submaxillary gland, but stops those going to the sublingual.

If the duct of the gland be connected with a mercurial manometer, it is found that when the chorda tympani is stimulated the pressure of secretion may exceed the blood pressure in the carotid, showing that the saliva is not formed by filtration.

When the perivascular sympathetics or when the sympathetic cord of the neck is stimulated, the blood-vessels of the gland constrict, and a flow of very viscous saliva takes place.

On the parotid gland the auriculo-temporal nerve (*Aur.T.*) acts in the same way as the chorda tympani acts on the other salivary glands. But stimulation of the fifth nerve above the otic ganglion, from which the auriculo-temporal takes origin, fails to produce any effect. On the other hand, stimulation of the glossopharyngeal nerve (*IX.*), as it comes off from the brain, acts upon the parotid gland. Since the glossopharyngeal is united by Jacobson's nerve (*J.N.*) to the small superficial petrosal which passes to the otic ganglion, it is obvious that the parotid fibres take this somewhat round-about course.

When the sympathetic fibres to the gland alone are stimulated, constriction of the blood-vessels but no flow of saliva occurs; but if, when the flow of watery saliva is being produced by stimulating the glossopharyngeal or Jacobson's nerve, the sympathetic fibres are stimulated, the amount of organic solids in the parotid saliva is very markedly increased.

The nerve fibres passing to the salivary glands are presided over by a centre in the medulla oblongata which acts reflexly. So long as this is intact, stimulation of the lingual or glossopharyngeal leads to a reflex flow of saliva. Other nerves may also act on this centre. Thus, gastric irritation, when it pro-

duces vomiting, causes a reflex stimulation of salivary secretion.

According to the investigations of Pavloff, the salivary glands react appropriately to different kinds of stimuli through their nervous mechanism. When sand or bitter or saline substances are put in a dog's mouth, a profuse secretion of very watery saliva ensues to wash them out. When flesh is given, a saliva rich in mucin is produced. When dry food is given, saliva is produced in greater quantity than when moist food is supplied. Pavloff further states that the sight of different kinds of food produces a flow of the kind of saliva which their presence in the mouth would produce.

ii. Swallowing.—The food after being masticated is collected on the surface of the tongue

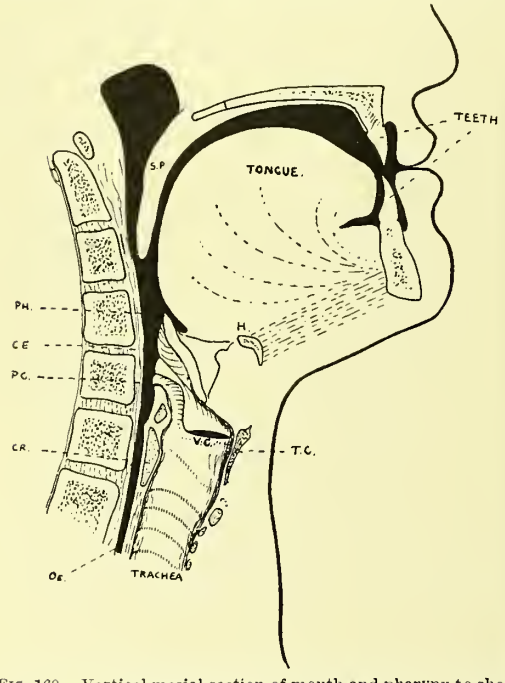


Fig. 160.—Vertical mesial section of mouth and pharynx to show how, in swallowing, the food slips along the back of the epiglottis. *PH.*, Pharynx; *C.E.*, cushion of epiglottis; *P.C.*, tip of arytenoid cartilages; *CR.*, cricoid cartilage.

by the voluntary action of the buccinators and other muscles, and then, the point of the tongue being pressed against the hard palate behind the teeth, by a contraction of the tongue passing from before backwards, the bolus of food is driven backwards. When the posterior part of the tongue is reached the act becomes purely reflex, and the food is forced through the pillars of the fauces into the pharynx. It is prevented from passing up into the posterior nares by the contraction of the palato-pharyngeus muscle, and of the levatores palati.

The larynx is pulled upwards as a whole by the stylo-hyoid and stylo-thyroid and the thyro-hyoid muscles, and the entrance of food is prevented by the closure of upper part of aperture. The arytenoid cartilages are pulled forward by the thyro-arytenoid muscles, and approximated by the arytenoidei, while a cushion on the posterior surface of the epiglottis becomes applied to their tips, forming a tri-radiate fissure or chink through which food cannot pass. The lateral crico-arytenoids also approximate the vocal cords, and close the glottis (Fig. 160).

The constrictors of the pharynx contract from above downwards, and force the food into the grasp of the œsophagus, and this by a slow peristaltic contraction sends the food onwards to the stomach. This peristalsis is abolished by section of the vagi, and it is generally not essential to swallowing. In swallowing liquids it is not brought into play, but the fluid is forced by the tongue down the relaxed œsophagus into the stomach.

The passage of the food into the stomach may be heard as a gurgling sound by applying a stethoscope to the right side of the spinal column, and any delay caused by a stricture may thus be determined. Normally the process takes five or six seconds.

In swallowing fluids two sounds are heard, one immediately, and one after about six seconds.

iii. Digestion in the Stomach. — Various opportunities have occurred and have been taken advantage of to study the interior of the human stomach during life. The best known investigation of the kind was undertaken by Dr Beaumont, a Canadian physician, on the person of St Martin, a backwoodsman, who had received a gunshot wound in the abdomen, which had left him with an opening through the front wall of his stomach. Dr Beaumont engaged St Martin as his servant, and made a prolonged and valuable study of the changes which take place in the viscus.

Within recent years the most important work

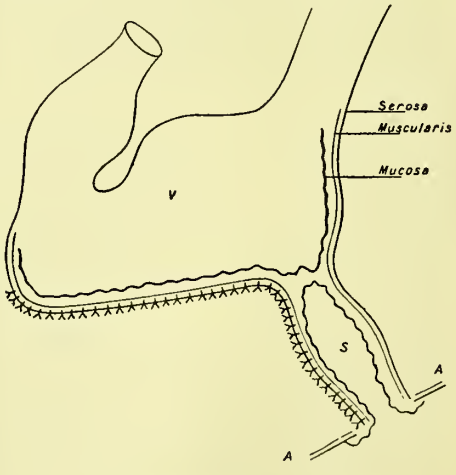


Fig. 161.—Diagram of Pavloff's pouch made on the stomach of a dog.

has been accomplished by Pavloff on dogs. His method is to make a small gastric pouch opening on the surface and separated from the rest

of the stomach (Fig. 161). This is done by cutting out a V-shaped piece along the great curvature, the apex being towards the pylorus and the base being left connected with the stomach wall. By a series of stitches the opening thus made in the stomach is closed up (top line of ΛΛs in Fig. 161), while the cut edges of the V-shaped flap are stitched together to form a tube. The one end of this is made to open upon the skin surface *A*, *A*, and by folding in the mucous membrane the deep end is isolated from the stomach. Thus a pouch is formed still connected with the nerves and vessels of the stomach, the condition of which represents the condition of the whole stomach.

The condition of the stomach varies greatly in fasting and after feeding.

A. *Stomach during Fasting*

The organ is collapsed, and the mucous membrane is thrown into large ridges. It is pale in colour because the blood-vessels are not dilated. Movements are not marked and the secretion is scanty, only a little mucus being formed on the surface of the lining membrane.

B. *Stomach after Feeding*

When food is taken, (1) the blood-vessels dilate, (2) a secretion is poured out, and (3) movements of the organ become more marked.

1. *Vascular Changes.*—The arterioles dilate, and the mucous membrane becomes bright red in colour. This is a reflex vaso-dilator effect, impulses passing up the vagus to a vaso-dilator centre in the medulla, and coming down the vagus from that centre. Section of the vagi prevents its onset.

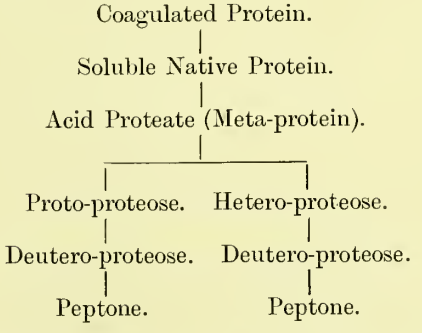
2. *Secretion.*—There is a free flow of gastric juice from all the glands in the mucous membrane.

(*a*) *Characters of Gastric Secretion.* — The gastric juice is a clear watery fluid, which is markedly acid from the presence of free hydrochloric acid. In the dog the free acid may amount to 0·2 per cent, but in man it is less abundant, and when the gastric juice is mixed with food the acid rapidly combines with alkalies and with proteins, and is no longer free. In addition to the HCl, small quantities of organic salts are present. Traces of proteins may also be demonstrated, and with these two enzymes are associated—one a proteolytic or protein-digesting enzyme, *pepsin*, the other a milk-curdling enzyme, *rennin*.

(*b*) *Course of Gastric Digestion*—(1) *Amylolytic Period.*—The action of the gastric juice does not at once become manifest. For half an hour after the food is swallowed the pytalin of the saliva goes on acting, and the various micro-organisms swallowed with the food grow and multiply, and thus there is a continuance of the conversion of starch to sugar which was started in the mouth, and at the same time the micro-organisms go on splitting the sugar to form lactic acid, which may thus be regarded as a normal constituent of the stomach during the first half-hour after a mixed meal.

(2) *Proteolytic Period.*—Before the amylolytic period is completed, the gastric juice has begun its special action on proteins. This may be readily studied by placing some coagulated protein in gastric juice, or in an extract of the mucous membrane of the stomach made with dilute hydrochloric acid, and keeping it at the temperature of the body. The protein swells, becomes transparent, and dissolves. The solution is coagulated on boiling—*a soluble native protein* has been formed. Very soon it is found that, if the soluble native protein is filtered off, the filtrate gives a precipitate on neutralising, showing that an *acid proteate* (*meta-protein*) has been produced. If the action is allowed to continue and the acid proteate precipitated and filtered off, it will be found that the filtrate gives a precipitate on saturating with common salt, showing that a *proto-proteose* has been formed. Along with this a certain amount of *hetero-proteose* is also formed. It is characterised by being precipitated on neutralisation and by being insoluble in distilled water. On filtering off these, the filtrate yields a precipitate on saturating with sulphate ammonium, indicating the formation of a *deutero-proteose*, and, if the filtrate from this be tested, the presence of a protein may be demonstrated. *Peptone* has been produced.

These changes may be represented in the following table :—



The process is one of breaking down a complex molecule into simpler molecules, probably with hydration.

The object of this was formerly supposed to be to allow of the diffusion of the protein in the form of peptone through the wall of the intestine. It is now known that absorption is not due to diffusion, and it is more probable that the change to the simplest protein molecule is a step to the more complete disintegration of the molecule which seems necessary before it can be built into the special protoplasm of the body of the particular animal.

On certain proteins and their derivatives the gastric juice has a special action. On *collagen*

the HCl acts slightly in converting it to gelatin. The gastric juice acts on *gelatin*, converting it to a gelatin peptone.

On *nucleo-proteins* it acts by digesting the protein part and leaving the *nuclein* undissolved.

Hæmoglobin is broken down into hæmatin and globin, and the latter is changed into peptone.

The *caseinogen calcium compound* of milk is first coagulated and then changed to peptone. The coagulation is brought about by the presence of the second enzyme of the gastric juice—rennin.

This may be separated from pepsin in various ways, and, unlike pepsin, it acts in a neutral medium.

The change set up by it seems to be due to a splitting of the soluble calcic compound of caseinogen which exists in milk into calcic paracasein, which is insoluble and is thrown down, and a small quantity of whey albumin which remains in solution. The nuclein part of the paracasein remains undigested.

The gastric juice contains an enzyme which splits fats into fatty acids and glycerine if they are in a very fine state of subdivision, as in milk, but it has no action on fats not so subdivided. When fats are contained in the protoplasm of cells, they are set free by the digestion of the protein covering.

On carbohydrates the free mineral acid of the gastric juice has a slight action at the body temperature, splitting the polysaccharids and disaccharids into monosaccharids.

(*c*) *Digestion of the Stomach Wall.*—When the wall of the stomach lies either in whole, as after the death of the animal, or in part, as when an artery is occluded or ligatured, the dead part is digested by the gastric juice and the wall of the stomach may be perforated. In the living condition a substance may be extracted from the mucous membrane which antagonises the action of pepsin and may be called antipepsin.

(*d*) *Antiseptic Action of the Gastric Juice.*—In virtue of the presence of free HCl the gastric juice has a marked action in inhibiting the growth of, or in killing bacteria. The bacillus of cholera is peculiarly susceptible, and a healthy condition of the stomach is thus a great safeguard against the disease. Other organisms, while they do not multiply in the stomach, pass on alive to the intestine, where they may again become active. When HCl is not formed in sufficient quantities to exist free in the stomach, the activity of these bacteria in the organ may lead to various decompositions and to many of the symptoms of dyspepsia.

(*e*) *Source of the Constituents of the Gastric Juice.*—The *hydrochloric acid* is formed at the cardiac end of the stomach. This may be shown by isolating a part of the stomach so that it opens on the surface. Since the parietal or oxyntic cells are confined to this portion of the stomach, it may be concluded that they are the producers of the acid. They manufacture it from the NaCl of the blood plasma. Probably the CO_2 liberated in the cells seizes on some of the Na and turns out HCl.

The *Pepsin* and *Rennin* are produced in the chief or peptic cells which line the glands both of the cardiac and pyloric parts of the stomach. During fasting granules are seen to accumulate in these cells, and when the stomach is active they are discharged. These granules are not pepsin but the forerunner of pepsin—pepsinogen.

(*f*) *Influence of Various Diets upon the Gastric Juice.*—This has been chiefly worked out by Pavloff on dogs with a gastric pouch (p. 60).

He finds that—(1) The amount of secretion depends upon the amount of food taken. (2) The amount and course of secretion varies with the kind of food taken. Thus, with flesh the secretion reaches its maximum at the end of one hour, persists for an hour and then rapidly falls, while with bread it reaches its maximum at the end of one hour, rapidly falls, but persists for a much longer period than in the case of flesh. (3) The digestive activity of the juices varies with the kind of food and with the course of digestion. It is higher and persists longer after a diet of bread, which is difficult to digest, than after a diet of flesh, which is more easily digested. (4) The percentage of acid does not vary markedly. When more acid is required more gastric juice is secreted. (5) The work done by the gastric glands is greater in the digestion of bread than in the digestion of flesh.

(*g*) *Nervous Mechanism of Gastric Secretion.*—It has been proved that in the dog the secretion of gastric juice can go on after the nerves to the stomach have been divided, and this has been ascribed to a reflex stimulation of the nerve plexus in the submucosa. But this mechanism plays a small part compared with the influences of the central nervous system through the vagus. Pavloff finds that, when the vagus is cut below the origin of the cardiac nerves so that they are not acted upon, and the animal left undisturbed for some days, stimulation of the nerve with a slowly interrupted induced current causes, after a long latent period of a minute or two, a flow of gastric juice.

This vagus action may be called into play either by the contact of suitable food with the mouth or by the sight of food. This, he demonstrated by making an œsophageal fistula in a dog with a gastric pouch, so that food put in the mouth escaped from the gullet and did not pass into the stomach (Fig. 162). Mere

mechanical or chemical stimulation of the mouth produces no effect, but the administration of meat produced it. The sight of food in a fasting dog produces after a latent period of five minutes a copious flow of gastric juice.

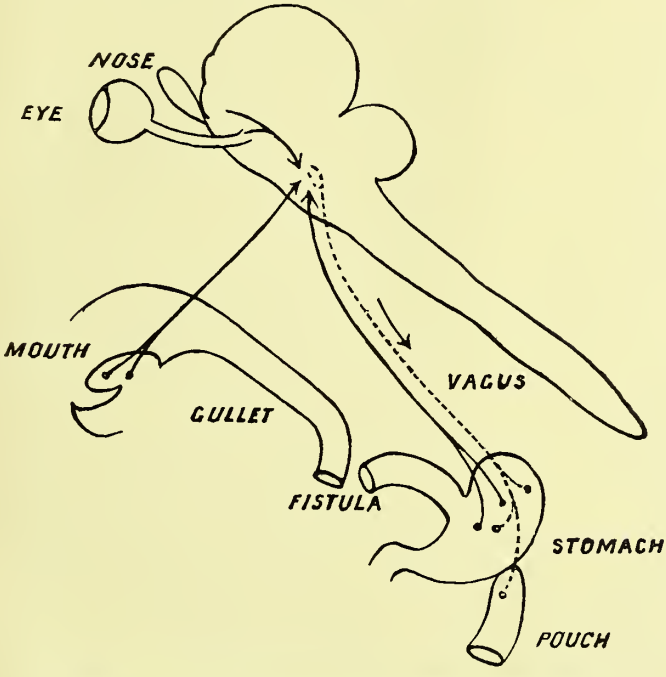


FIG. 162.—To show the nervous mechanism of gastric secretion and how it is reflexly induced through various ingoing channels.

Pavloff calls this "psychic" stimulation. It is an example of how the "distance receptor" in the eye reflexly brings about an appropriate reaction—just as the "non-distance receptor" in the wall of the stomach under certain stimuli brings about an appropriate reaction. It is somewhat rash of a physiologist who can know nothing of the relation of the psychic state to the actions with which it is associated to affirm, as Pavloff does, that the psychic change is causal.

There is some evidence that the formation of gastric juice is also influenced by the action of a chemical substance produced in the mucous membrane of the pyloric end of the stomach. It has been found that the injection into the blood stream of an extract of this membrane made by boiling with acid or peptone causes a production of gastric juice. In all probability the initial secretion of gastric juice is dependent on the nervous mechanism, and the secondary secretion, when food is in the stomach, on the action of this substance. In the case of the pancreas such a chemical stimulant plays a very important part (p. 82). Such substances have been named Hormones.

3. *Movements of the Stomach.*—These have been studied by feeding an animal with food containing bismuth, and then applying X-rays, which are intercepted by the coating of bismuth, so that a shadow picture of the shape of the stomach is given (Fig. 163).

It is found that, soon after food is taken, a constriction forms about the middle of the stomach and slowly passes on towards the pylorus. Another constriction forms and follows the first, and thus the pyloric part of the stomach is set into active peristalsis. The cardiac fundus acts as a reservoir, and, by a steady contraction, presses the gastric contents into the more active pylorus, so that at the end of gastric digestion it is completely emptied. The pylorus is closed by the strong sphincter muscle, which, however, relaxes from time to time during gastric digestion to allow the escape of the more fluid contents of the stomach into the intestine. These openings are at first slight and transitory, but as time goes on they become more marked and more frequent, and when gastric digestion is complete —usually at the end of five or six hours—the sphincter is completely relaxed and allows the stomach to be emptied. The openings are regulated by a local nervous mechanism which is brought into play by the escape of the acid gastric contents into the duodenum. This leads to an immediate closure of the pylorus, which does not again open till the contents of the duodenum have been neutralised by the alkaline secretions which are poured into it. The rate of passage from the stomach of various kinds of food has

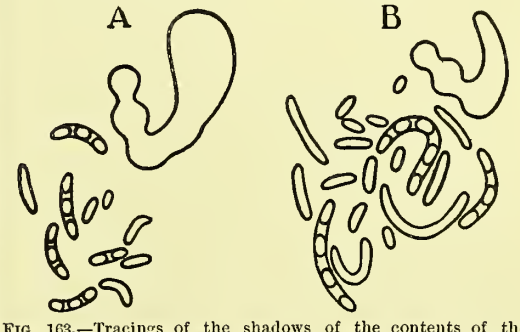


FIG. 163.—Tracings of the shadows of the contents of the stomach and intestine of a cat two hours after feeding (A) with boiled lean beef, and (B) with boiled rice to show the more rapid emptying of the stomach after the carbohydrate food. The small divisions of the food in some of the intestinal loops represent the process of rhythmic segmentation (*see* p. 70). (Cannon.)

been studied by feeding cats with equal amounts of each kind of food mixed with bismuth, and then, by X-rays, getting the outline of the contents of the small intestine at different periods. Carbohydrates were found to pass on most rapidly and fats most slowly (Fig. 163).

Unless the contents of the stomach are very fluid these movements do not produce a very great mixing of the food taken. This separation of the food into layers according to the time at which it was taken is very well marked in the horse, but has also been demonstrated in the dog (Fig. 164).

Nervous Mechanism of Gastric Movements.—Even after the section of all the gastric nerves, movements of the stomach may be observed, but the mechanism of these movements has not been fully studied. The action of the vagus and sympathetic fibres is complicated, and their influence on the wall of the stomach and the sphincters requires further investigation. Speaking generally, the vagus seems to increase the movements, while the sympathetic fibres check them. The vagus when stimulated generally causes inhibition of the cardiac sphincter and contraction of the pylorus, but the result is not constant.

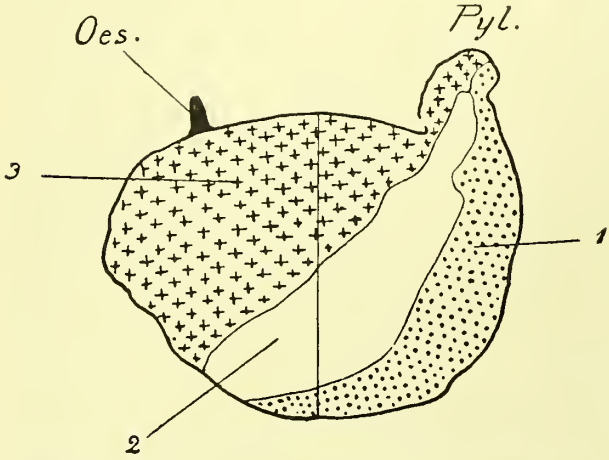


FIG. 164.—Stomach of a dog fed successively with three different foods to show the absence of mixing. (Scheunert.)

Absorption from the Stomach.—By ligation of the pyloric end, it has been found that the stomach plays a very small part in the absorption of food. Its chief function is to act as a reservoir.

Probably the antiseptic action of its secretion is of considerable importance. While it plays a certain part in digestion its action is by no means indispensable, for it has been removed in animals and in men without disturbance of the health.

Vomiting.—Sometimes the stomach is emptied upwards through the gullet instead of downwards through the pylorus. This act of vomiting is generally a reflex one, resulting from irritation of the gastric mucous membrane, and more rarely from stimulation of other nerves. It is a reaction to nocuous stimuli.

Usually the act is preceded by a feeling of nausea and by a free secretion of saliva. In vomiting, the glottis is closed, and, after a forced inspiratory effort by which air is drawn down into the gullet, a forced and spasmodic expiration presses on the stomach, while at the same time the cardiac sphincter is relaxed through the action of the vagus, and the contents of the stomach are sent upwards. They are at first prevented from passing into the nares by the contraction of the muscles of the soft palate; but, as the act continues, these muscles are overcome, and the vomited matter escapes through mouth and nose. The wall of the stomach also seems to act, but its action is non-essential, since vomiting may be produced in an animal in which a bladder has been inserted in place of the stomach.

The centre which presides over the act is in the medulla oblongata, and while it is usually reflexly called into action, it may be stimulated directly by such drugs as apomorphine.

iv. Intestinal Digestion.—After being subjected to gastric digestion the food is generally reduced to a semi-fluid, grey, pultaceous condition of strongly acid reaction known as *chyme*, and in this condition it enters the duodenum.

Here it meets three different secretions :—

Pancreatic secretion.
Intestinal secretion.
Bile.

A. *Pancreatic Secretion*

The secretion of the pancreas may, in the dog, be procured by making either a temporary or a permanent fistula. In the former case the duct is exposed, and a cannula fastened in it; in the latter the duct is made to open on the surface of the abdomen, a small piece of the intestinal wall with the mucous membrane round the opening of the duct being stitched to the abdominal opening.

1. *Characters and Composition.*—When obtained immediately from a temporary fistula, the pancreatic juice is a clear, slimy fluid, with a specific gravity of 1030 or less and an alkaline reaction. It contains an abundance of a native protein having the characters of a globulin, and the alkalinity is probably due to sodium carbonate and disodium phosphate. From a permanent fistula a more abundant flow of more watery secretion may be collected.

2. *Action.*—Closely associated with the protein, and precipitated by alcohol along with it, are the enzymes upon which the action of the pancreatic juice depends.

1*st. A Proteolytic Enzyme—Trypsin.*—This, in a weakly alkaline or neutral fluid, converts native proteins into peptones, and then breaks these peptones into simpler non-protein bodies.

The pancreatic juice brings about this breaking down of protein in stages. It does not

cause solid proteins to swell up, but simply erodes them away. Fibrin and similar bodies first pass into the condition of *soluble native proteins* and then into *deutero-proteose*, while boiled egg white appears at once to yield *deutero-proteose*. The deutero-proteose is then changed into peptone, and part of that peptone is then split into a series of bodies which no longer give the biuret test. These consist chiefly of the component amido-acids, of which the most important are leucin and tyrosin, and of ammonia compounds (*see* vol. vii. p. 491).

Amido-acetic acid linked to skatol—tryptophane—is also split off, and if chlorine water is added to a pancreatic digestion which has proceeded for a long time, a rose-red colour is struck (*see* p. 88).

On nucleo-proteins trypsin acts by digesting the protein and dissolving the nucleic acid so that it can be absorbed.

On collagen and elastin trypsin has little action; but on gelatin it acts as upon proteins.

2nd. An Amylolytic Enzyme—Amylopsin or *Diastase.*—This acts in the same way as ptyalin, but more powerfully, converting a certain part of the maltose into dextrose. It acts best in a faintly acid medium.

3rd. A Fat-splitting Enzyme—Lipase.—This is the most easily destroyed and the most difficult to separate of the zymins. It breaks the fats into their component glycerin and fatty acids. The fatty acids link with the alkalies which are present to form soaps, and in this form, or dissolved as free fatty acids in the bile, they are absorbed. But the formation of soaps also assists the digestion of fats by reducing them to a state of finely divided particles, an *emulsion*, upon which the lipase can act more freely. This process of emulsification is assisted by the presence of protein in the pancreatic juice and also by the presence of bile.

That these enzymes are independent of one another is shown by many facts.

(1) Diastase does not appear till about a month after birth.

(2) Diastase is taken up by dry glycerin, while trypsin is not.

(3) Trypsin may be precipitated and separated by shaking with collodion.

(4) Trypsin acts in 0·01 per cent ammonia while diastase does not.

(5) The proportion of the zymins varies with the character of the diet.

This is well shown by experiments carried out in Pavloff's laboratory upon dogs with pancreatic fistulæ. The effects of diets of milk, bread, and flesh were compared, in each case the amount of the food given containing the same amount of nitrogen (protein). The total quantity of ferment unit is got by multiplying the quantity of juice in c.cm. by the strength of the juice, determined by ascertaining how much of the substance is digested by unit of the secretion in unit of time. The following table and Fig. 165 indicate the results obtained:—

Diet.	Quantity of Enzyme.		
	Proteolytic.	Amylolytic.	Fat-splitting.
Bread, 250 grm. .	1978	1601	800
Milk, 600 c.c. . .	1085	432	4334
Flesh, 100 grm. . .	1502	648	3600

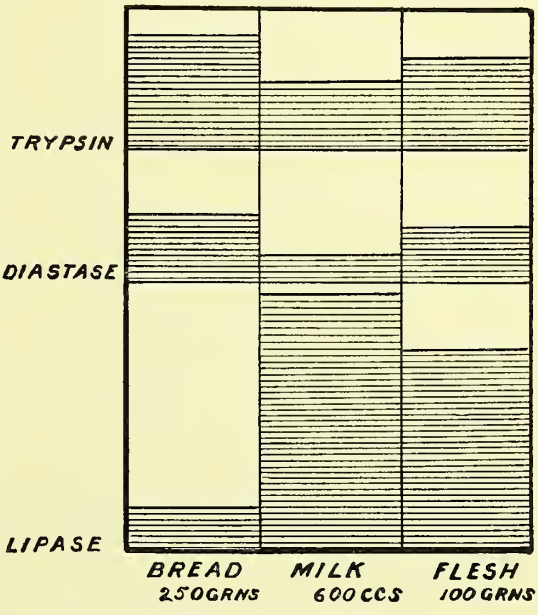


FIG. 165.—To show the relative amounts of the three enzymes of the pancreatic juice formed on different diets.

Bread contains a protein difficult of digestion, plenty of starch, and little fat. Milk contains an easily digested protein, and plenty of fat, but no starch; while flesh contains a comparatively easily digested protein, no starch, and a moderate amount of fat. The first food causes a copious production of trypsin and diastase, and little lipase. The second causes the production of less trypsin, little diastase, but most lipase. The last causes a moderate production of trypsin, little diastase, and a comparatively large amount of lipase.

As to the mode of production of these enzymes, it is known that trypsin is not formed as such in the cells, for the secretion direct from the acini has no tryptic action. A forerunner of trypsin—trypsinogen—is produced, and this changes into trypsin after it is secreted. The intestinal secretion contains something of the nature of an enzyme which has been termed enterokinase, which has the power of bringing about this change.

It is doubtful whether the pancreatic secretion contains any true rennin, although it produces a modified clotting of milk, under certain conditions.

3. *Physiology of Pancreatic Secretion.*—The secretion of pancreatic juice is not constant, but is induced when the acid chyme passes into the duodenum. This occurs even when all the nerves to the intestine have been cut, and it appears from the investigations of Starling to be due to the formation of a material which has been called secretin, in the epithelium lining the intestine, under the influence of an acid. This is absorbed, and, on being carried to the pancreas, stimulates it to secrete. It has been shown that the injection into the blood of an extract, made with dilute hydrochloric acid, of the lining membrane of the upper part of the small intestine, leads to a flow of pancreatic juice. This secretin is not destroyed by boiling, and is soluble in strong alcohol. It is therefore not of the nature of an enzyme.

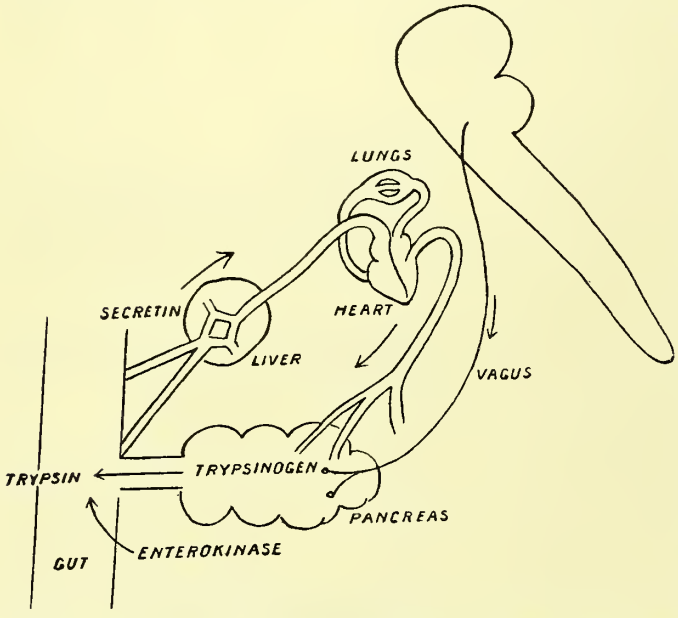


FIG. 166.—To show the mode of action of secretin and the action of the vagus nerve on the secretion of the pancreas and the activation of trypsinogen by enterokinase.

But while secretin seems to play so important a rôle, it has been found that stimulation of the vagus nerve, after a latent period of two minutes, increases pancreatic secretion, so that it must be concluded that the secretion of the fluid is, to a certain extent, under the control of the nervous system.

The influence of the pancreas in the general metabolism will be considered later (p. 84).

B. *Secretion of the Intestinal Wall (Succus Entericus)*

This is formed in the Lieberkühn's follicles of the intestine, and it may be procured by cutting the intestine across at two points, bringing each end of the intermediate piece to the surface, and connecting together the ends from which this piece has been taken away so as to make a continuous tube. On mechanically irritating the mucous membrane, a pale yellow clear fluid is secreted, which contains native proteins and mucin, and is alkaline in reaction from the presence of sodium carbonate.

Action.—The succus entericus contains: (1) An enzyme which splits some disaccharids, as maltose and cane sugar, into monosaccharids, but does not seem to act on lactose. (2) In the intestine of animals taking milk a special zymin, lactase, which splits milk sugar. (3) Erepsin, an enzyme which seems to act more powerfully than trypsin in splitting peptones into their component non-protein crystalline constituents such as the di-amido acids and mon-amido acids, *e.g.* leucin and tyrosin. The object of this is not at present fully understood. It may be that the nitrogen of the protein is largely treated as a waste product and thrown off. But feeding experiments on dogs and rats seem to show that these non-protein derivatives of proteins can be retained and built into the protoplasm of the animal. Vernon has shown that a similar proteolytic enzyme is widely distributed in the tissues, being specially abundant in the kidney. (4) Enterokinase—a zymin which, acting on trypsinogen, converts it into active trypsin (p. 65).

Mechanism of Secretion.—The taking of food leads to a flow of intestinal secretion which reaches its maximum in about three hours; and this flow is much greater from the upper part of the bowel than from the lower. There is some evidence that the injection of secretin calls forth this secretion, and according to some observers, the injection of succus entericus into the circulation acts in the same way.

As regards the action of nerves very little is known. It has been found that, when the intestine is ligatured in three places so as to form two closed sacs, if the nerves to one of these be divided, it becomes filled with a clear fluid closely resembling lymph. The dilatation of the blood-vessels may account for this without secretion being implicated.

C. *Bile*

1. *Characters and Composition.*—The bile is the secretion of the liver, and it may be procured for examination—(*a*) From the gall bladder, or (*b*) from the bile passages by making a fistula into them. Bile which has been in the gall bladder is richer in solids than bile taken directly from the ducts, because water is absorbed by the walls of the bladder and the bile thus becomes concentrated.

Analyses of gall bladder bile thus give no information as to the composition of the bile when formed. In several cases, where surgeons have produced biliary fistulæ, opportunities have occurred of procuring the bile directly from the ducts during life in man.

Such bile has a somewhat orange-brown colour, and is more or less viscous, but not nearly so viscous as bile taken from the gall bladder. It has a specific gravity of almost 1005, while gall bladder bile has a specific gravity of about 1030. Its reaction is slightly alkaline, and it has a characteristic smell.

It contains about 2 per cent of solids, of which more than half are organic.

Bile Salts.—The most abundant solids are the salts of the bile acids. In man the most important is sodium glycocholate. Sodium taurocholate occurs in small amounts. These salts are readily prepared from an alcoholic solution of dried bile by the addition of water-free ether, which makes them separate out as crystals.

Glycocholic acid splits into glycin, amido-acetic acid—$H_2N.CH_2.CO.OH$, and a body of unknown constitution, cholalic acid, $C_{24}H_{40}O_5$.

Taurocholic acid yields amido-ethane-sulphuric acid or taurin, $H_2N.CH_2CO.SO_2OH$—a molecule closely resembling amido-acetic acid linked to sulphuric acid and cholalic acid.

Since both acids contain nitrogen they must be derived from proteins. That they are formed in the liver and not merely excreted by it, is shown by the fact that, while they accumulate in the blood if the bile duct is ligatured, they do not appear if the liver is excluded from the circulation.

Action of Bile Salts.—(1) The bile salts are solvents of fats and fatty acids, and they thus assist in the digestion and absorption of fats. When bile is excluded from the intestines no less than 30 per cent of the fats of the food may escape absorption and appear in the fæces. When this is the case, as in jaundice from obstruction of the bile duct, the fæces have a characteristic white or grey appearance from the abundance of fat.

(2) These salts keep cholesterin in solution.

(3) They lower the surface tension of solutions, and in this way they may bring the fat particles into more intimate contact with the mucous membrane.

(4) While the salts have no action on proteins, free taurocholic acid precipitates native proteins and acid proteate. In the human intestine this is an action of no importance.

(5) These salts are powerful hæmolytic agents, and rapidly dissolve hæmoglobin out of the erythrocytes.

Bile Pigments.—These amount to only about 0·2 per cent of the bile. In human bile the chief pigment is an orange-brown, iron-free substance, *bilirubin*, $C_{32}H_{36}N_4O_6$, while in the bile of herbivora, *biliverdin*, a green pigment somewhat more oxidised than bilirubin, $C_{32}H_{36}N_4O_8$, is more abundant. By further oxidation with nitrous acid, other pigments—blue, red, and yellow — are produced, and this is used as a test for the presence of bile pigments (Gmelin's test).

The pigments are closely allied to hæmatoporphyrin and hæmatoidin (*see* p. 6), and they are derived from hæmoglobin. Their amount is greatly increased when hæmoglobin is set free or injected into the blood. That they are formed in the liver is shown by the fact that, when the liver is excluded from the circulation, the injection of hæmoglobin does not cause their formation.

The liver has the property of excreting not only these pigments formed by itself, but also other pigments. Thus the liver of the dog can secrete the characteristic pigment of sheep's bile when this is injected into its blood.

Cholesterin is a monatomic alcohol—$C_{26}H_{43}OH$—which occurs free in small amounts in the bile. It is very insoluble and is kept in solution by the salts of the bile acids. It readily crystallises in rhombic plates, generally with a notch out of the corner. On account of its insolubility, when it is in excess in the bile or when the bile salts are decreased, it may form concretions or biliary calculi — gall stones — which may accumulate in the gall bladder and may get caught in the bile passages, obstructing the flow of bile and leading to its absorption throughout the system. Jaundice is thus produced. When these stones are forced along the bile passages as a result of muscular contraction, intense agony—biliary colic—may be produced. When they are passed by the rectum, their nature is readily demonstrated by breaking them up in a mortar, dissolving in hot alcohol, and allowing the solution to cool, when the characteristic crystals separate out. The source of the cholesterin of the bile is not definitely known. It is not an excretion of cholesterin formed elsewhere, because the injection of cholesterin does not lead to an increase in the amount in the bile. According to Naunyn's observations it is most abundant in cases of inflammation of the bile passages, and he therefore thinks that it is formed by the breaking down of the epithelium lining these ducts.

Fats and Lecithin.—The true fats and the phosphorus containing lecithin are present in small amounts in the bile, and apparently they are derived from the fats of the liver cells, and they may be increased in amount by the administration of fatty food.

Nucleo-protein and Mucin. — The bile owes its viscosity to the presence of a mucin-like body, which, however, does not yield sugar on boiling with an acid and which contains phosphorus. It is precipitated by acetic acid,

but the precipitate is soluble in excess. It is therefore a nucleo-protein. In some animals a certain amount of mucin is also present.

Inorganic Constituents.—The most abundant salt is calcium phosphate. Phosphate of iron is present in traces. Sodium carbonate, calcium carbonate, and sodium chloride are the other chief salts.

2. *Flow of Bile.* — The bile, when secreted by the liver cells, may accumulate in the bile passages and gall bladder to be expelled under the influence of the contraction of the muscles of the ducts or of the pressure of the abdominal muscles upon the liver. The flow of bile into the intestine thus depends upon — 1st, The secretion of bile; 2nd, the expulsion of bile from the bile passages. It is exceedingly difficult to separate the action of these two factors. The flow of bile in the human subject has now been studied in several cases in which the surgeon has had to make a fistula into the gall bladder through which all the bile secreted escaped and could be collected.

The flow of bile begins in intra-uterine life before the twelfth week, and it continues without intermission throughout the whole of life, even during very prolonged fasts.

The taking of food increases the flow of bile, and the extent to which it is increased depends largely on the kind of food taken. In the dog a protein meal has the most marked effect, a fatty meal has a marked effect, and a carbohydrate meal hardly any effect. The increased flow of bile following the taking of food does not reach its maximum till six or nine hours after the food is taken, and some observers have found that the period of maximum flow is even further prolonged.

Pavloff found in dogs, in which a biliary fistula had been made leaving the opening of the bile duct in the mucous membrane of the intestine, that an *expulsion* of bile follows the taking of food and the secretion of pancreatic juice, and Starling finds that the flow of bile is increased by the injection of secretin. It thus tends to run parallel with the flow of pancreatic juice.

When the individual is taking a liberal diet, the secretion of bile appears to be greater than when the diet is low.

In fever there is a very marked fall in the secretion, the fluid flowing from a fistula becoming colourless and almost devoid of bile salts and pigments.

Certain drugs markedly modify the formation of bile—the salts of the bile acids stimulating the liver to form more solids and to secrete more water, the salicylates acting in much the same way, and all drugs which cause hæmolysis — *i.e.* the solution of the pigment of the erythrocytes — producing an increased formation of bile pigments.

Influence of Nerves upon the Flow of Bile.—(*a*) *Expulsion of Bile.*—There is good evidence that nerve fibres pass to the muscles of the bile passages and that they may cause an expulsion of bile by stimulating them to contract.

(*b*) *Secretion of Bile.*—There is no evidence that nerve fibres act directly upon the secretion of bile. This appears to be governed by the nature of the material brought to the liver by the blood and by the activity of the liver cells. It is an example of function regulated by chemical substances rather than by a nerve mechanism.

3. *Mode of Formation of Bile.*—It has been seen that the bile salts and pigments are actually formed in the liver cells, and there is good evidence that the water of the bile is not a mere transudation but is the product of the living activity of these cells. The pressure under which bile is secreted may be determined by fixing a cannula in the bile duct or in a biliary fistula and connecting it with a water manometer. In man the pressure is as much as 20 to 30 mm. Hg, while the pressure in the portal vein of the dog is only 7 to 16 mm. Hg.

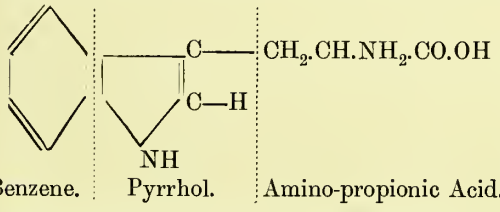
4. *Nature and Functions of Bile.*—Bile is not a secretion of direct importance in digestion. It has practically no action on proteins or carbohydrates, and its action on fats is merely that of a solvent, and possibly by its action on the surface tension of the intestinal contents. Pavloff maintains that it activates the lipase of the pancreatic juice, and others have found that it increases the activity of trypsin and diastase. It may thus be considered as an adjuvant to the action of pancreatic juice. Its secretion in relationship to food does not indicate that it plays an active part in digestion. It is formed during intra-uterine life and during fasting, and it is produced many hours after food is taken, when digestive secretions are no longer of use in the alimentary canal. Digestion can go on quite well without the presence of bile in the intestine, except that the fats are not so well absorbed. The composition of bile strongly suggests that it is a waste product. The pigment is the result of the decomposition of hæmoglobin and the acids are the result of protein disintegration.

All these facts seem to indicate that bile is the medium by which the waste products of hepatic metabolism are eliminated, just as the waste products of the body generally are eliminated by the kidneys.

Bacterial Action in the Alimentary Canal.—With the food and saliva numerous micro-organisms of very diverse character are swallowed. It has been suggested that the leucocytes formed in the lymphoid tissue at the back of the mouth and pharynx attack and destroy such organisms, but so far definite proof of this is not forthcoming. When the food is swallowed, the micro-organisms multiply for some time in the warm moist stomach, and

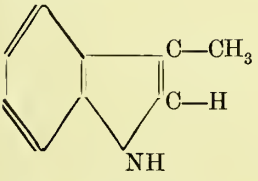
certain of them, by splitting sugars, form lactic and sometimes acetic acid. But when sufficient gastric juice is poured out for the hydrochloric acid to exist free, the growth of micro-organisms is inhibited, and some of them, at least, are killed. Others pass on into the intestine, and, as the acid in the chyme becomes neutralised, the acid-forming organisms begin to grow, and, by splitting the sugars, form lactic or acetic acid, and render the contents of the small intestine slightly acid. Towards the end of the small intestine, and more especially in the large intestine, the alkaline secretions have neutralised these acids, and in the alkaline material so produced the putrefactive organisms begin to flourish and to attack any protein which is not absorbed—splitting it up and forming among other substances a series of aromatic bodies, of which the chief are indol, skatol, and phenol.

This splitting probably occurs through the liberation of tryptophane—in which amido-propionic acid is linked to a pyrrhol-benzene.

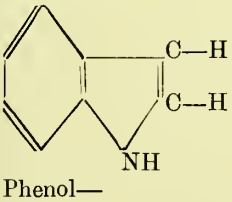


Benzene. Pyrrhol. Amino-propionic Acid.

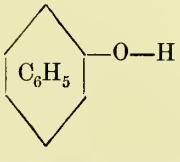
By the breaking down of the amino-propionic acid, skatol—



is formed, and by the removal of the methyl, indol is produced—



Phenol—



is a further stage of disintegration.

By taking embryo guinea-pigs at full time from the uterus and keeping them with aseptic precautions, it has been shown that the absence of micro-organisms from the intestine does not interfere with digestion.

The most important and abundant organism present in the intestinal tract is the *bacillus coli communis*, which has a certain power of splitting proteins and a marked action in producing acids from sugars. Its presence in water is generally indicative of sewage contamination.

Fate of the Digestive Secretions.—1. *Water.*—Although it is impossible to state accurately the average amount of the various digestive secretions poured into the alimentary canal each day, it must be very considerable, probably not far short of 3000 c.cms., or something considerably more than one-half of the whole volume of the blood. Only a small amount of this is given off in the fæces, and hence the greater part must be reabsorbed. There is thus a constant circulation between the blood and the alimentary canal, or what may be called an *entero-hæmal circulation.* One portion of this is particularly important. The blood-vessels of the intestine pass to the liver, and many substances, when absorbed into the blood stream, are again excreted in the bile and thus are prevented from reaching the general circulation. Among these substances are the salts of the bile acids and their derivatives, many alkaloids such as curarine, and in all probability a set of animal alkaloids called *ptomaines* formed by putrefactive decomposition of proteins in the gut. If, from disturbances in the functions of the liver, these are allowed to pass through that organ, the feelings of lassitude and discomfort which are associated with intestinal dyspepsia are produced. The liver thus forms a protective barrier to the ingress of certain poisons.

2. *Enzymes.*—*Ptyalin* appears to be destroyed in the stomach by the hydrochloric acid. *Pepsin* is probably partly destroyed in the intestine, but it seems also to be absorbed and excreted in the urine; for, on the addition of hydrochloric acid, the urine has a peptic action on proteins. *Trypsin* appears to be destroyed in the alimentary canal; but the fate of the other pancreatic enzymes and of the enzymes of the succus entericus is unknown.

3. *Bile Constituents.*—1. The *bile salts* are partly reabsorbed from special parts of the small intestine — sodium glycocholate being taken up in the jejunum and taurocholate in the ileum. The acids of these salts are also partly broken up. The glycocholic acid yields amido-acetic acid, which is absorbed and passes to the liver to be excreted as urea; while the taurocholic acid yields amido-isethionic acid which goes to the liver and yields urea and probably sulphuric acid. The fate of the cholalic acid is not known, but it is supposed to be excrcted in the fæces. 2. The *pigments* undergo a change and lose their power of giving Gmelin's reaction. They appear in the fæces as what may be called *stercobilin.* It is probably formed by reduction of bilirubin in the intestines as the result of the action of micro-organisms. 3. The *cholesterin* is passed out in the fæces.

Fæces.—The materials not absorbed from the intestine, whether these are derived from the food or from the alimentary canal, are thrown off from the rectum as the fæces. In *fasting animals* these are passed at long intervals, and consist of mucin, shed epithelium, the various products of the bile constituents, and inorganic salts. In *feeding animals* the amount and character of the fæces depend largely upon the amount and character of the food, and upon the bacteria which are growing in the large intestine. The unabsorbed material, as it passes down the large intestine, becomes inspissated from the absorption of water, but, if much undigested matter is present, water may also be added, and the consistence of the fæces may thus be varied. In the average condition they contain about 70 or 80 per cent of water. The colour is normally brown, from the hæmatin of the flesh eaten, while the sulphide of iron formed by the splitting of the hæmatin compounds in the intestine may make them darker in colour. On a milk diet they are light yellow in colour, and if a large excess of fatty food is taken, or if fat is not absorbed, as in jaundice, they become clay-coloured. The derivatives of the bile pigments play but a small part in colouring the fæces. In infants, before bacteria are introduced and begin to exert their reducing action, the fæces may be green from the presence of unaltered biliverdin.

The reaction of the fæces varies. Usually the outside of the mass is alkaline from the alkaline secretion of the intestine, while the inside is acid from the free fatty acids and other acids formed by the action of such acid-forming bacteria as the bacillus coli communis. The amount of solid fæces depends on the amount of food—a fairly average amount per diem is 150 grms. of dried solids. On a vegetable diet, from the presence of undigested cellulose, the amount is very much greater.

The solids of the fæces of a feeding animal consist of the same constituents as the fæces in a fasting animal, with the addition of all the undigested constituents of the food—elastic and white fibrous tissue, remains of muscle fibres, often fat, and the earthy soaps of the fatty acids, and, when a vegetable diet is taken, the cellulose of the vegetable cells, and frequently starch. The cellulose, by stimulating the intestine, is a valuable natural purgative.

The odour is due to the presence of aromatic bodies such as indol and skatol.

Meconium is the name given to the first fæces passed by the child after birth. It is greenish-black in colour, and consists of inspissated bile and shed epithelium from the intestine.

Movements of the Intestine.—These are of two kinds—myogenic and peristaltic. The *myogenic* movements are slight rhythmic contractions which pass rapidly along the intestine, and are insufficient to drive on the contents, but are probably of use in churning and mixing them. By feeding with food mixed with bismuth, and employing X-rays, Cannon finds that the contents of the small intestine get broken up into small segments. This is possibly due to these myogenic movements (Fig. 163, p. 63). These movements occur when all the nerves have been divided, and when the ganglia in the intestinal walls have been poisoned with nicotine, and they are therefore due to the muscle fibres alone.

The *peristaltic* movements are much more complex and powerful. They consist of a constriction of the muscles, which seems to be excited by the passage of the food, and may be caused by inserting a bolus of cotton-wool covered with vaseline. Starting at the upper end of the intestine, they pass slowly downwards. In front of the contraction the muscular fibres are relaxed, and thus the contracting part drives its contents into the relaxed part below. These peristaltic movements go on after the nerves to the gut are cut, but they are stopped when the ganglia in the wall of the intestine are poisoned with nicotine. It has therefore been concluded that the nerve ganglia in the intestinal wall form a local reflex mechanism, which is stimulated by the presence of foreign matter in the intestine, and which brings about the co-ordinated contraction and relaxation, which together constitute a true peristalsis.

But while peristalsis is thus independent of the central nervous system, it is nevertheless controlled by it. The splanchnic nerves inhibit, while the vagus to the small intestine and upper part of the large gut, and the nervi erigentes to the lower part of the large gut, are augmentor nerves, increasing the peristalsis. Stimulation of the sympathetic fibres which inhibit the peristalsis causes contraction of the sphincter between the small and large intestine.

As the contents of the small intestine are forced through the ileo-cæcal valve, the large intestine relaxes to receive them, and then a series of contractions passing from below upwards—an anti-peristalsis—sets in by which the contents are very thoroughly churned. Afterwards they are forced downwards by tonic peristaltic waves.

The intestinal movements are inhibited by emotions.

Defæcation.—By the peristalsis of the intestine, the matter not absorbed from the wall of the gut is forced down and accumulates in the part of the rectum which passes horizontally forward to end in the vertical anal canal. It is prevented from escaping into this by the sharp fold which the last part of the bowel makes, and by the contraction of the strong sphincter ani muscle.

Defæcation depends primarily on the intestinal peristalsis, without which it cannot be performed. When fæces accumulate in the

rectum, the mucous membrane is stimulated, and impulses are sent up to inhibit a centre in the lumbar region of the cord which keeps the sphincter ani contracted, and the sphincter is relaxed and the escape of fæces made possible. In some diseases of the cord this centre is stimulated and cannot be inhibited, and thus defæcation is interfered with, while in other diseases, when this centre has been destroyed, the sphincter does not contract, and fæces may escape continuously.

Normally the act of defæcation is partly voluntary and partly involuntary. The voluntary part of the act consists in closing the glottis and making a forced expiration so as to press upon the contents of the abdomen, while at the same time the perineal muscles are relaxed and the rectum straightened, and thus the contents are allowed to pass into the anal canal. The act is completed by the emptying of the canal by the contraction of the levatores ani muscles.

III. Absorption of Food.—1. *State in which Food leaves the Alimentary Canal.*—The *carbohydrates* generally leave the alimentary canal as monosaccharides; but some resist the action of digestion more than others. Lactose seems to be broken down in the intestine only when the special *lactase* is present in the succus entericus, but in all cases it is broken down before it reaches the liver. Cane sugar when taken in large exeess may also be absorbed, and it is then excreted by the kidneys.

The *proteins* are absorbed as peptones, possibly as proteoses, and as the amino-acids and other crystalline compounds formed by the action of trypsin and erepsin (vol. vii., p. 490). Native proteins may be absorbed unchanged from the lower bowel, since it has been found that when egg white is injected into an isolated part of the rectum it disappears to a very considerable extent.

The *fats* are chiefly absorbed as soaps and as fatty acids.

2. *Mode of Absorption of Food.*—That absorption is not due merely to a process of ordinary diffusion or osmosis is clearly indicated by many facts.

(1) Heidenhain has shown that absorption of water from the intestine takes place much more rapidly than diffusion through a dead membrane.

(2) The relative rate of absorption of different substances does not follow the laws of diffusion. Grübler's peptone passes more easily through the intestine than glucose, but glucose passes more readily through parchment paper, while sodium sulphate, which is more diffusible than glucose, is absorbed much less readily. Again, as shown by Reid, an animal can absorb its own serum under conditions in which filtration into blood capillaries or lacteals is excluded. In such a case osmosis cannot play a part. Absorption is stopped or diminished when the epithelium is removed, injured, or poisoned with fluoride of sodium, in spite of the fact that this must increase the facilities for osmosis and filtration.

3. *Channels of Absorption.*—There are two channels of absorption from the alimentary canal (*see* Fig. 115, p. 10)—the veins which run together to form the portal vein of the liver, and the lymphatics which run in the mesentery and, after passing through some lymph glands, enter the *receptaculum chyli* in front of the vertebral column. From this, the great lymph vessel, the thoracic duct, leads up to the junction of the subclavian and innominate veins, and pours its contents into the blood stream. The lymph formed in the liver also passes into the thoracic duct.

(1) *Proteins.*—Peptones and the further products of their digestion are formed from proteins in digestion, but they seem to undergo a change in the intestinal wall before passing to the tissues, since they are not found in the blood. That in some altered condition they leave the intestine by the blood and not by the lymph is shown by the fact that their absorption is not interfered with by ligature of the thoracic duct.

During the digestion of proteins the number of leucocytes is enormously increased, sometimes to more than double their previous number, and in all probability it is they which carry the products of digestion from the intestine. According to the observations of Pohl, the leucocytes are derived from the lymph tissue in the intestinal wall, but more recent experiments tend to show that they come from the bone marrow, being probably attracted to the intestine by a positive chemiotaxis. By breaking down in the blood stream they probably set free the proteins for use in the tissues.

When an excess of proteins is taken in the food, it is broken down in the lining membrane of the gut, and the nitrogen is rapidly excreted in the urine as urea, and thus the entrance of an excess of nitrogen to the tissues is prevented. Its non-nitrogenous part remains available as a source of energy.

It has been pointed out that gastric juice does not dissolve the nucleo-proteins, but that the pancreatic juice does so. Phosphorus is undoubtedly absorbed in organic combination, but the mode of absorption and the channels by which it passes from the intestine have not been investigated.

(2) *Carbohydrates.*—Although the chief monosaccharid formed in digestion is dextrose, others are also produced—lævulose from cane sugar and galactose from milk sugar. All these are absorbed in solution, and are carried away in the blood of the portal vein.

(3) *Fats.*—After being split up into the component acids and glycerin, fats pass, as soluble soaps or as fatty acids soluble in the bile, through the borders of the intestinal epithelium.

Here they appear to be again converted into fats by a synthesis of the acid with glycerin. Fine fatty particles are found to make their appearance in the cells at some distance from the free margin and to increase in size. A similar synthesis occurs even when free fatty acids are given, so the cells must be capable of producing the necessary glycerin to combine with the acids. The fats are sent on from the cells, through the lymph tissue of the villi, into the central lymph vessels, and thus on, through the thoracic duct, to the blood stream. Unlike the proteins and carbohydrates, they are not carried directly to the liver.

IV. Fate of the Food Absorbed

The food absorbed may be—

(*A*) used immediately as a source of energy, for (1) the Construction or Reconstruction of Tissues; (2) the Production of Mechanical Work; (3) the Production of Heat;

Or (*B*) it may be stored for future use in the body.

The processes of construction and repair of the tissues and the production of mechanical work have already been considered (vol. vii., p. 492 *et seq.*), and the production of heat and the regulation of temperature may now be dealt with.

I. *Production of Heat and Regulation of Temperature*

1. Production of Heat.—A. *Muscle.*—The production of heat in muscle has been already studied (vol. vii. p. 511). It has been shown that muscle, from its great bulk and constant activity, is the main source of heat in the body. Not only may it be demonstrated that the temperature of contracting muscle rises, but it has been found that the temperature of blood coming from the muscles is slightly higher than that of blood going to them. Muscular exercise raises the temperature of the body. The shivering fit which is induced by exposure to cold is really a reflex reaction by which heat production is increased. Drugs which interfere with muscular contraction, such as curare, diminish the temperature, and young animals, before their muscular tissues become active, have a low temperature unless kept in a warm atmosphere.

B. *Glands.*—Wherever chemical change goes on in protoplasm, heat is liberated. Therefore in glands during activity a certain amount of heat is produced. But the production in them is trivial when compared with the production in muscle. During the period of active digestion the temperature of the blood coming from the liver may be nearly 1° C. higher than that of the blood going to the organ. The liver, alone among glandular structures, contributes an appreciable amount of heat to the body, since the amount of blood passing through the organ is large, and thus a considerable amount of heat is derived from it.

C. *Brain.* — Some physiologists have maintained that the fact that the temperature of the brain rises during cerebral activity indicates that the chemical changes going on are sufficient to yield a certain amount of heat. But it is more probable that the rise of temperature is due to the increased flow of blood through the organ, since a study of the gases in the blood coming from the brain gives no indication of any marked increase of chemical change during periods of increased cerebral action.

2. Regulation of Temperature.—Since heat is constantly being produced, the temperature of the body would tend to rise higher and higher, were there not some arrangement by which just as much heat is eliminated as is produced, and by which the temperature is thus kept constant.

Elimination of Heat.—Heat is got rid of by three channels. A. *Skin.*—Since the body is generally warmer than the surrounding air, heat is constantly lost by *conduction*, *convection*, and *radiation*, and the extent of this loss depends mainly upon the difference between the temperature of the body and that of the air. Radiation plays the most important part when a person is sitting quiet in still air; conduction and convection when the exchange of air over the surface is rapid. The temperature of the skin is increased when, from dilatation of the cutaneous vessels, more blood is brought to the surface, and conversely it is lowered by constriction of these vessels. The influence of variations in the temperature of the air is generally minimised in man by the covering of clothes, and in animals by the covering of fur or feathers, which retains a stationary layer of air at about 25° to 30° C. over the skin. It has been calculated that in man over 70 per cent of all the heat is lost by conduction and radiation.

The loss of heat by radiation may be determined by finding to what extent a thin metal grill fixed at a definite distance from the surface of the body is heated. This can be done by determining the change produced in its electric conductivity.

By the *evaporation of sweat*, heat is rendered latent, and is taken from the body, which is thus cooled just as the hand may be cooled by allowing ether to evaporate upon it. If the amount of sweat vaporised is known, it is possible to calculate the amount of heat removed from the body in this way. The loss is comparatively small—only about 14 per cent of the whole. The extent depends upon the rapidity with which evaporation goes on, and this is governed by the amount of sweat secreted, and by the dryness and temperature of the atmosphere. Thus a warm dry climate is better borne than one which is warm and moist, since in the former the loss of heat by evapora-

tion is so much greater. Of the various factors increasing sweat secretion, heat is probably the most important.

Since the temperature of the skin is governed by the state of the cutaneous vessels and the amount of sweat produced by the state of the sweat glands, and since both of these are under the control of the nervous system, the elimination of heat from the skin is presided over by a nervous mechanism.

B. *Respiratory Passages.*—By conduction and radiation and by evaporation from the respiratory passages, about 10 per cent of the heat is got rid of in man. In the dog and some other animals, the proportion of heat eliminated in this way is considerably greater.

C. *Urine and Fæces.*—Since these are warmer than the surrounding air, a certain amount of heat is lost through them. The amount is small—something less than 2 per cent of the whole.

Temperature.—In all higher animals, the loss of heat and the production of heat are so nicely balanced that the temperature of the body remains fairly constant under all conditions. If an extra amount of heat is produced, say in muscular exercise, it is at once eliminated by the skin; and, if the body is exposed to a low temperature, loss of heat is rapidly checked by contraction of the cutaneous vessels and diminished activity of the sweat glands.

Since heat is constantly being given off, the temperature at the surface of the body is always lower than the temperature in the interior. The temperature of the rectum may be taken as a measure of the internal temperature.

The mean daily temperature of a healthy man is:

	°C.	°F.
Rectum . . .	37·2	98·96
Axilla . . .	36·9	98·45
Mouth . . .	36·87	98·36

But the temperature varies throughout the course of life.

	°C.
Infant . . .	37·5
Under 25 years. .	37·2
About 40 . . .	37·1
Old Age . . .	37·2 to 37·5

It also varies throughout the course of the twenty-four hours, and since this is a matter of great importance in medicine, it has been very carefully studied by many observers. The difference is not more than 1° C. It is lowest in the early morning and highest in the afternoon (Fig. 167).

Under all normal conditions the temperature of man undergoes only small variations, because the balance between production of heat and elimination of heat is so well maintained. But under abnormal conditions the balance is frequently upset. Thus severe muscular work causes a temporary rise of temperature, because heat elimination does not quite keep pace with heat production. Exposure to very high temperatures may cause a slight rise of temperature,

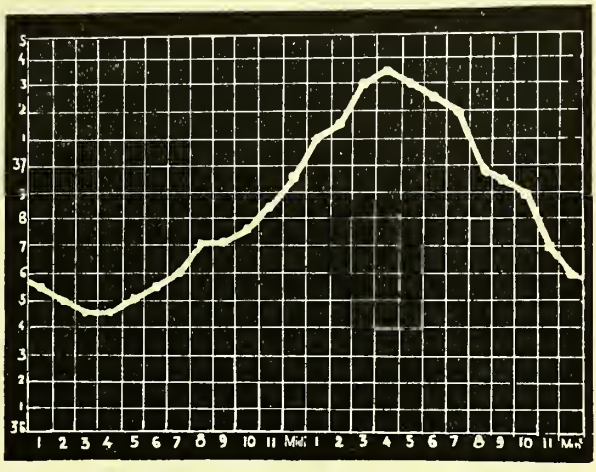


FIG. 167.—Chart of the daily variation of temperature in the normal human subject in degrees centigrade. (Richet.)

while exposure to excessive cold may cause a slight fall; but, unless, in the case of those unable to use their muscles—*e.g.* in those suffering from alcoholic poisoning—the change is small.

Although the normal variation of temperature is so small, life may be sustained when the temperature falls for a time to about 25° C., or rises to nearly 43° C. Cases are recorded in which it has even risen to 46·6° C. without death supervening.

While the higher "warm-blooded animals," mammals and birds, maintain a constant temperature, the lower vertebrates, "cold-blooded animals," reptiles, amphibia, and fishes, do not do so, and their temperature varies with that of the surrounding medium.

But even in mammals the mechanism for the regulation of temperature is not absolutely perfect, and in every species of animal there is a limit to the power of adjustment.

Mammals which hibernate become for the time "cold-blooded animals," and lose their power of regulating their temperature.

The regulation of temperature may be effected either by modifying heat production, or by altering the rate of elimination.

Heat production is voluntarily modified when muscular exercise is taken during exposure to cold, and involuntary when muscles are set in action by a shivering fit. There is, however, no evidence of the existence of a special nervous mechanism presiding over heat production in muscle.

But it is not so much by changes in the rate of heat production, as by alteration in heat

elimination through the skin, that the temperature is kept uniform. The nerves to the cutaneous vessels, and to the sweat glands, are the great controllers of temperature. It is through failure of this mechanism under the action of the toxins of micro-organisms that heat elimination is diminished and the temperature is raised in fevers.

It is not necessary to assume that there is a special heat regulating nervous mechanism, since the nervous arrangements presiding over the vessels and glands of the skin are capable of immediately responding to change of condition calling for their intervention.

II. *Storage of Surplus Food*

A. Since bulk for bulk fat has more than twice the energy value of proteins or carbohydrates, it is an advantage to store surplus food as fat.

This storage takes place chiefly in three situations: (1) fatty tissue; (2) muscle; (3) liver.

1. *In Fatty Tissues.*—In most mammals the chief storage of surplus food is in the fatty tissues.

That the fat of the food can be stored in them is shown by the fact that the administration of large amounts of fats different from those of the body leads to their appearance in those tissues.

Fats are also formed from the carbohydrates of the food. Feeding experiments upon pigs and other animals, carried out in this country by Laws and Gilbert, have definitely proved that sugary foods are changed to fat in the body and stored in that form. The following may be given as an example of such experiments. Two young pigs of a litter were taken, and one was killed and analysed. The other was fed for weeks on maize, the amount eaten being weighed and the excretion of nitrogen by the pig being determined. The animal was then killed and analysed, and it was found that the fat gained was more than could be produced from the fat and protein of the food eaten. It must therefore have been formed from the carbohydrates.

The evidence that fats may be formed from the *proteins* of the food is conflicting. In the ripening of cheese it is undoubted that under the influence of micro-organism proteins are changed to fats, and in all probability the same thing occurs in the formation of the fatty adipocere in the muscles of the dead body during putrefaction. At one time it was supposed that under the influence of such poisons as phosphorus the proteins of the cells of the mammalian tissues are changed to fat. But careful chemical examination has shown that the so-called fatty degeneration is due to accumulation of already existing fats in the affected organs. Voit fed dogs on lean beef, and found that, while all the nitrogen was discharged from the body, the carbon was retained, and he concluded that it was retained as fat. But he failed to recognise that even lean flesh contains both fat and glycogen from which the fat can be formed. At present we have no direct evidence that the fats of the body are formed from proteins.

2. *In the Liver.*—The liver is a storehouse of carbohydrates and fats (p. 75). Lecithin is always present in the liver, even in prolonged fasting.

3. *In Muscle.*—Some animals, as the salmon, store fats within their muscle fibres; but in mammals such a storage is limited in amount.

B. Proteins may, to a small extent, be stored in muscle, especially after a fast or a prolonged illness. But in the healthy mammal it is difficult to get such a storage, except in athletic training, where the muscles may be enormously increased by the building up of the protein derivatives of the food into their protoplasm.

C. Carbohydrates are stored to a small extent in the liver and in the muscle.

III. *The Liver in relationship to Absorbed Food and to the General Metabolism*

The liver develops as a couple of diverticula from the embryonic gut, and is thus primarily a digestive gland, and in invertebrates it remains as a part of the intestine both structurally and functionally. But in mammals, early in fœtal life, it comes to have important relationships with the blood going to nourish the body from the placenta. The vein bringing the blood from the mother breaks up into a series of capillaries in the young liver, and in these capillaries the development of the cells of the blood goes on for a considerable time. Soon the liver begins to secrete bile, while animal starch and fat begin to accumulate in its cells. Gradually the formation of blood cells stops, and the mass of liver cells become larger in proportion to the capillaries. As the fœtal intestine develops, the vein bringing blood from it—the portal vein—opens into the capillary network of the liver, so that, when at birth the supply of nourishment from the placenta is stopped, the liver is still associated with the blood bringing nutrient material to the tissues.

1. *Relation to Carbohydrates — Glycogenic Function.* — Claude Bernard discovered that sugar is formed in the liver. This formation of sugar goes on throughout life, and on account of this constant supply the amount of sugar in the blood does not diminish, even when an animal undergoes a prolonged fast. In starvation there are only two possible sources of this glucose — the fats and the proteins of the tissues. There is no conclusive evidence that fats can be changed to sugar in the liver, although it is difficult to explain the large amount of sugar which is sometimes excreted

in phloridzin poisoning, unless it is formed from fats. That it is not all formed from proteins is shown by the fact that the sugar which appears is sometimes greater than could be produced by the proteins broken down, as indicated by the output of nitrogen (p. 76).

That proteins are a source of sugary substances is shown by the amount of sugar which is produced by an animal rendered diabetic by removal of its pancreas and fed exclusively on proteins. It is therefore probable that in starvation the proteins of the body are broken down and their non-nitrogenous part changed to sugar.

But not only does the liver manufacture sugar for the tissues in starvation, but, when the supply of sugar is in excess of the demands of the tissues, it stores it as a form of starch—glycogen (*see* p. 54)—and gives it out as sugar as that substance is required. On a carbohydrate diet the accumulation of glycogen in the liver is very great; but even on a protein diet, in dogs at least, a smaller accumulation takes place. The observation that the various monosaccharids are all stored as the same form of glycogen shows that they must first be assimilated by the liver protoplasm and then converted to glycogen, the process being one of synthesis.

The way in which glycogen is again changed to sugar is doubtful. The fact that the liver, after treatment with alcohol, can change glycogen to glucose, has induced some physiologists to believe that it is by an enzyme that this conversion goes on during life. But it has been shown (1) that the injection of methylene blue, which poisons protoplasm but does not interfere with the action of enzymes, checks the conversion, and (2) that stimulating the splanchnic nerves going to the liver increases the conversion of glycogen without increasing the amylolytic enzyme in the liver and blood. It is therefore probable that the conversion results from chemical changes in the protoplasm which are controlled by the nerves of the liver.

If more sugar is taken than the liver can deal with, it passes on into the general circulation, and is excreted in the urine. Every individual has a certain power of oxidising and storing sugar, and most persons can dispose of about 200 grms. at a time. But the carbohydrate capacity varies greatly, and even in the same individual it is different under different conditions. When the glycogen stored in the liver is changed to glucose more quickly than is required by the tissues, the glucose may be to a certain extent again stored in the muscles as glycogen, or it may accumulate in the blood and be excreted in the urine (glycosuria). This condition is seen when the posterior part of the floor of the fourth ventricle in a rabbit is punctured. If glycogen be abundant in the liver, glycosuria results, the stimulation of the nervous system producing a too rapid conversion of the glycogen.

Another way in which sugar may be made to appear in the urine is by injecting phloridzin. Under the influence of this drug the sugar in the blood is not increased. It must be concluded that it acts by causing the kidneys to excrete glucose too rapidly, so that it is not available for the tissues. But even when carbohydrates are withheld and cleared out of the body, phloridzin causes glycosuria. Hence the kidneys must be made to form glucose from the protein of the blood plasma.

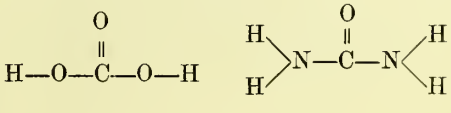
The injection of large doses of extract of the suprarenal bodies causes a glycosuria with an increase of sugar in the blood; but so far it is not known whether the condition is one of increased production or of diminished utilisation of sugar (p. 82).

Removal of the pancreas also causes glycæmia and glycosuria (p. 84).

2. *Relation to Fats.*—Although the fats are not carried directly to the liver, as are proteins and carbohydrates, they are stored in large amounts in the liver of some animals—*e.g.* the cod among fishes and the cat among mammals. Animals which have little power of storing fat throughout the muscles and other tissues generally, seem to have a marked capacity for accumulating it in the liver. Even in starvation the fats do not disappear from the liver, and throughout all conditions of life a fairly constant amount of lecithin, a phosphorus and nitrogen containing fat (*see* vol. vii. p. 517), is present in the liver cells. Lecithin, in the yolk of the egg, is an intermediate stage in the formation of the more complex nucleins of living cells, and the formation of lecithin in the liver by the synthesis of glycerin, fatty acids, phosphoric acid, and cholin is probably a first step in the construction of these nucleins. If this be so, the fat of the liver must play an important part in retaining and fixing phosphorus in the body.

3. *Relation to Proteins.* — Along with the intestinal wall, the liver regulates the supply of proteins to the body. A study of the chemical changes in muscle has shown that the waste of protein is normally small in amount, and that a great part of the nitrogen is capable of being used again if a supply of oxygen and carbonaceous material is forthcoming (*see* vol. vii. p. 514). Hence the demand for nitrogen in the muscles is small, and for this reason, apparently, any excess of protein in the food is decomposed, either by trypsin and erepsin or by the intestinal wall, into simple nitrogenous compounds, which are changed into urea in the liver.

Urea, the chief waste substance excreted in the urine, is the bi-amide of carbonic acid.



It contains 46·6 per cent of nitrogen. It is a white substance crystallising in long prisms. It is very soluble in water and alcohol—insoluble in ether. With nitric and oxalic acids it forms insoluble crystalline salts. It is readily decomposed into nitrogen, carbon dioxide, and water by nitrous acid and by sodium hypobromite in excess of soda.

Urea is chiefly formed in the Liver.—That it is not produced in the kidneys is shown by the following facts:—(1) When these organs are excised, urea accumulates in the blood. (2) When ammonium carbonate is added to blood artificially circulated through the kidney of an animal just killed, no urea is formed.

That it is not formed in the muscles is shown —(1) By the absence of a definite increase in urea formation during muscular activity; (2) by the fact that when blood containing ammonium carbonate is streamed through muscles, urea is not produced.

That it is formed in the liver is indicated—(1) By the fact that when an ammonium salt such as the carbonate dissolved in blood is streamed through the organ, it is changed to urea; (2) by the observation that when the liver is cut out of the circulation, the urea in the urine rapidly diminishes, and ammonia and lactic acid take its place.

The exclusion of the liver from the circulation in mammals is difficult, because, when the portal vein is ligatured, the blood returning to the heart tends to accumulate in the great veins of the abdomen. But this difficulty has been overcome by Eck, who devised a method of connecting the portal vein with the inferior vena cava, and finally occluding the portal vein, and of thus allowing the blood to return from the abdomen to the heart.

Source of Urea.—Urea is produced from the decomposition products of proteins of the food and tissues (*see* p. 7). The manner in which excess of protein in the *food* is broken down into ammonia compounds in the intestine and sent to the liver has been already considered (p. 71). But the fact that even in starvation urea is produced seems to indicate that the initial stages of decomposition of proteins may go on elsewhere than in the intestinal wall. The fate of hæmoglobin tends to show that the whole process may be conducted in the liver cells. When hæmoglobin is set free from the corpuscles, the nitrogen of its protein part is changed to urea, while the pigment part is deprived of its iron and excreted as bilirubin. Whether the proteins of muscle and other tissues are thus directly dealt with, or whether the initial stages of decomposition go on outside the liver, is not known. But the wide distribution of erepsin through the tissues may indicate that the initial splitting of the protein goes on in them.

The nitrogen excreted is not all in the form of urea, but some is combined in ammonia salts, in uric acid and other purin bodies (*see* p. 94), and in creatinin. In the mammalian body ammonia and the purin bodies can be changed into urea, and it is probable that the small amounts of these substances which appear in the urine have simply escaped this conversion. Certain drugs (alcohol, sulphonal, etc.) and toxins (diphtheria) markedly decrease their conversion into urea and so increase their quantity in the urine. Although urea may be prepared from creatin, there is no evidence that the process goes on in the body. Creatin yields the creatinin of the urine.

It is probable that after the nitrogenous portion of the protein molecule is split off and got rid of, the liver has the further power of turning the non-nitrogenous part into sugar and either sending it to the tissues or storing it as glycogen.

Summary of the Functions of Liver.—The functions of the liver may be briefly summarised as follows:—(1) It regulates the supply of glucose to the body (*a*) by manufacturing it from proteins when the supply of carbohydrates is insufficient, and (*b*) by storing it as glycogen when the supply of carbohydrates is in excess, and giving it off afterwards as required. (2) Along with the intestinal wall it regulates the supply of proteins to the body, by decomposing any excess, and giving off the nitrogen as urea, etc. (3) It regulates, in many animals at least, the supply of fat to the body by storing any excess. (4) It regulates the number of erythrocytes by getting rid of waste hæmoglobin and retaining the iron for further use (*see* p. 6). (5) From the part it plays in the entero-hepatic circulation, it protects the body against certain poisons by excreting them in the bile (*see* p. 67).

V. General Metabolism

Having considered how the food is digested and absorbed, and how it is then either stored or at once used (*a*) for building up and repairing the tissues, or (*b*) as a source of energy, *the rate at which the various chemical changes go on and the factors modifying them* may be dealt with.

The changes in the two great constituents of the body—proteins and fats—have to be separately studied.

1. Method of Investigating.—A. *Protein Metabolism.*—The amount of protein used in the body is readily calculated from the amount of nitrogen excreted, since, under normal conditions, unless nitrogen in some unusual combination is being taken, it is derived entirely from the proteins in the body. Proteins contain 16 per cent of nitrogen, and hence each grm. of nitrogen excreted is derived from 6·25 grms. of protein.

The nitrogen is almost entirely excreted in the urine. Only a small amount escapes by the bowels and skin, and hence only when very

accurate observations are desired is it necessary to analyse the fæces and sweat.

B. *Metabolism of Fats.* — Proteins contain nearly three and a half times as much carbon as nitrogen, and hence, when they are broken down, for each grm. of nitrogen excreted, 3·4 grms. of carbon are given off.

The carbon is chiefly excreted from the lungs as carbon dioxide, and in this form it may be collected and estimated.

Any excess of carbon excreted, over the 3·4 times the amount of nitrogen given off, must be derived from the fats of the body or from the fats and carbohydrates taken in the food. Any carbon retained in the body, apart from that in proteins, is stored ultimately as fat. Since carbon constitutes 76·5 per cent of fats, the amount of fat is calculated by multiplying the carbon by 1·3.

The following tabular example of an investigation of the metabolism may be given :—

	Intake in Grams.			Output.	
		C.	N.	C.	N.
Proteins . .	100	54	16	...	...
Fats . . .	100	76	...	...	...
Carbohydrates .	400	200	...	...	...
		330	16	300	14

Two grms. of nitrogen are retained as protein ; that is, 2 × 6·25 = 12·5 grms. of protein—are being daily laid on. Thirty grms. of carbon are also retained in the body, and of this 3·4 × 2 = 6·8 grms. are combined with the nitrogen in the protein. The remainder, 23·2 grms., go to form fats, the amount of which is 23·2 × 1·3 = 30·16 grms. of fat.

2. Metabolism during Fasting.—When the usual supply of energy in the food is cut off, the animal liberates the energy required by oxidising its own stored material and its tissues. This is shown by the fact that the animal loses weight and goes on excreting carbon dioxide, urea, and the other waste products of the activity of the tissues.

Several prolonged fasts have been undertaken by men, and, in one or two of these, careful observations have been made by physiologists. It has been found that during the first day or two of a fast, the individual goes on using proteins and fats at something like the same rate as he did while taking food, but that gradually he uses less and less protein each day. This is well shown in the case of Succi, who underwent a fast of thirty days.

Day of Fast.	Protein used in Grms.	Fat used.
1st	104	Not estimated.
10th	51	170
20th	33	170
29th	31	163

It is from the stored fats that the energy is chiefly derived, and the result of this is that before death the fats of the body are largely used up. The protein-containing tissues waste more slowly and waste at different rates, the less essential being used up more rapidly than the more essential, which, in fact, live upon the former. In cats deprived of food till death supervened the heart and central nervous system had hardly lost weight ; the bones, pancreas, lungs, intestines, and skin each had lost between 10 to 20 per cent of their weight, the kidneys, blood, and muscles between 20 to 30, and the liver and spleen between 50 to 70.

The *rate of waste* during a fast depends upon the amount of energy required, and it is therefore increased by muscular work and by exposure to cold. When a man is kept quiet and warm and supplied with water, a fast of thirty days may in some cases be borne without injury.

3. Effect of Feeding.—When food is given to a fasting animal or man, the first effect is to increase the rate of wasting by calling into action the muscles and glands concerned in digestion. The result is an immediate increase in the excretion of nitrogen and carbon, indicating an increased breaking down of proteins and fats. It has been found that in the dog a diet of white bread and butter increased the metabolic processes by an amount equivalent to about 10 per cent of the energy value of the diet. For this reason, to give an animal which is fasting a diet containing just the amount of nitrogen and of carbon which the animal is excreting, will not at once stop the loss of weight. Suppose, for instance, that to a fasting animal using daily 30 grms. of the proteins and 160 grms. of the fats of his body, a diet containing these amounts is given, the disintegration of proteins and of fats will at once rise, say, to 50 grms. of protein and 280 of fat. Thus the result will be that, instead of his losing 30 grms. of protein, he will lose only 20 grms. per diem, and instead of 160 grms. of fat, only 120 grms. But, if the diet is sufficient to supply the energy required, in a few days the intake and output will balance, and the individual is then said to be in metabolic equilibrium, and he neither gains nor loses weight. The following table and Fig. 168 give an idea of how this adjustment of the metabolism is reached :—

Day.	Intake.		Disintegrated.		Waste diminished to.	
	Protein.	Fat.	Protein.	Fat.	Protein.	Fat.
1	0	0	30	160	...	...
2	30	160	50	280	20	120
3	30	160	40	240	10	60
4	30	160	30	200	0	40
5	30	160	30	160	0	0
6	30	160	30	160	0	0

If the amount of food be further increased, a small proportion of the proteins and a larger proportion of the fats are retained, and weight is gained. As already indicated, the power of storing proteins is generally small (*see* p. 74).

Protein Diet.—Proteins contain all the chemical elements required for the building and repair of the tissues, and from the complexity of their molecules they also supply latent energy. It is therefore theoretically possible for an animal to sustain life on proteins, and certain animals can be fed exclusively upon them. Thus Pflüger kept a dog for many months upon a purely protein diet without injury to its health. But to supply the necessary energy in

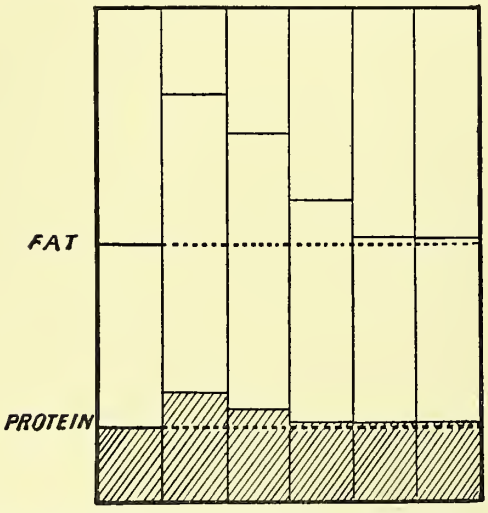


FIG. 168.—To show the effects on the metabolism of proteins and fats of feeding a fasting animal. The continuous horizontal lines indicate the amount of material metabolised, the broken horizontal lines the amount taken. The differences between the levels of these indicate the amount of protein and of fats of the animal body which are metabolised. The first column represents the condition in fasting—the succeeding columns the intake and output each day when food is given.

proteins alone requires the consumption of excessively large quantities. For a man to get the energy equivalent to 3000 calories—a very moderate expenditure per diem—he would have to eat more than seven times the usual amount of proteins. Further, it has been shown that, when large quantities are taken, the greater portion is broken up in the intestinal wall and formed into urea by the liver and excreted by the kidney, and thus excessive work is thrown upon these excretory organs. While these organs usually form and excrete about 33 grms. of urea per diem, on such a diet they would have to deal with no less than 231 grms.

It is therefore not advantageous to adopt a too purely protein diet. The great use of proteins is as muscle-builders. When the muscles are in a state of constant activity they have a certain power of laying on protein as they grow. Hence the value of proteins in muscular training.

Gelatin, although undergoing digestion and absorption like the proteins, is not available as a muscle-builder. Its sole use is as an energy yielder, and in this respect it has a value equal to the proteins.

Carbohydrate Diet. — Carbohydrates are of equal value with proteins as a source of energy, but they contain no nitrogen, and they are not available alone for building up and repairing the protoplasm of muscles and other tissues. Carbohydrates alone will not support life, but when added to proteins they enable the animal to do with smaller quantities of the latter. They are thus sometimes termed *protein sparers*. Their use in diminishing the consumption of proteins is, however, strictly limited.

Fat Diet.—Fats, like carbohydrates, will not support life, because they cannot be used for building up protoplasm; but, like carbohydrates, they are a source of energy, and they have more than twice the energy value of proteins or of carbohydrates (p. 53). They are thus *protein sparers*. But experiment has shown that, in spite of their higher energy value, they have not the same power as carbohydrates of sparing proteins, since their digestion and absorption is more difficult. Thus while theoretically 1 grm. of carbohydrate yields 4·1 calories of energy, 1 grm. taken in the food, since it is not all absorbed and used, yields 4·03 calories of energy. But fat, which theoretically yields 9·3 on account of its less availability, yields in the body only 8·93 calories. That is, while carbohydrates yield 97 per cent of their energy, as determined by the calorimeter, fats yield only 92 per cent of their energy.

When once absorbed, fats are iso-dynamic with the equivalent amount of carbohydrates. This has been determined by replacing the carbohydrates in a diet with the equivalent amount of fat.

A knowledge of the part played by proteins, carbohydrates, and fats in the animal body is the groundwork of the study of *dietetics*.

VI. Dietetics

The object of dietetics is to fit the supply of matter and energy in the food to the requirement of the body under different conditions.

The great essentials of a diet capable of maintaining health are:—

1*st*. That it should be capable of digestion, absorption, and assimilation, *i.e.* that it should be available.

2*nd*. That it should supply the energy required.

3*rd*. That it should contain sufficient proteins to make good the waste of these substances.

I. The Availability of the Food.—While proteins, carbohydrates, and fats in the alimentary canal undergo the changes already described, they are frequently, when taken in the food, in an unfavourable state for the action of digestive juices. Thus, while the proteins of

flesh are exposed to the gastric and pancreatic secretion, the proteins of many vegetables, unless carefully prepared, cooked, and masticated, are protected and escape digestion and absorption. The same may be said of the crude starch of vegetables. The digestibility and value as an article of diet of the cellulose and allied substances in plants is an important question at present requiring further investigation.

The fats of vegetables are also generally in a less favourable state for digestion than the fats of animals.

When once digested there seems to be no difference in the *absorbability* and *assimilability* of the proximate principles of vegetables and animals. A given weight of vegetable protein or vegetable fat may be substituted for the same quantity of animal protein or fat.

As a result of the differences in digestibility, the *availability* of food-stuffs varies. The following table serves to show some of these variations:—

Availability of Food-Stuffs

	Per Cent Absorbed.		
	Protein.	Fat.	Carbohydrate.
Flesh . .	98	98	...
Egg . .	97	98	...
Milk . .	89–99	96	100
Bread . .	88	...	98
Potatoes (boiled)	73	...	98
Carrots (raw) .	61	...	82

In an ordinary mixed diet the availability of the three proximate principles is about—

Protein.	Fat.	Carbohydrate.
92	95	97

And hence in such a diet the available energy value is less than the energy value of the principles of the food as determined in the calorimeter.

Thus in a mixed diet the *available* energy value of 1 grm. of—

Protein is 4·05 calories.
Fat is 8·93 ,,
Carbohydrates is 4·03 ,,

The availability of almost any article of food varies with the state of the teeth and the digestive organs of the individual, with the manner in which it is eaten—whether leisurely or too rapidly and without proper mastication, and with the manner in which it is prepared. For example, the mode of manufacture of the flour used in bread-making has a very marked influence upon the amount digested and absorbed. In vegetable foods especially the thoroughness of the cooking has a most important influence on the availability of their constituents.

By *assimilation* is meant the taking of the food constituents from the blood by the muscle and other tissues so that they may be used. Apparently the only substances which can be freely assimilated and used by the tissues are the three proximate principles of the food, although alcohol, and possibly some other similar substances, may be utilised to a small extent.

II. The Energy Requirements.—The energy requirements of the individual, which the food has to meet, vary with the age and size and with the mode of life.

Size.—Other things being equal, a large man requires more energy and more protein than a small man. For this reason the energy requirements are sometimes stated as per unit of weight, but it is more convenient to take as the standard an adult man of average weight, say 65 kgs. It must further be remembered that the smaller the animal the greater the surface in proportion to its weight; and hence the greater the loss of heat per unit of weight. For this reason alone small animals and children require more energy per unit of weight than larger animals or older people.

Age.—In children the metabolism is more active than in adults. They are more constantly in motion, they require energy and material to build up their tissues, and, as just stated, the loss of heat per unit of weight is greater than in their elders. Weight for weight, a child thus requires a greater supply of energy than a man. The following results, based upon observations on diets recorded by Camerer, illustrate this:—

Age.	Weight in Kilos.	Energy used per Kilo in Calories.	Total Energy in Calories.
4	14	91·3	1280
12	30	57·7	1730
30	66	42·4	2800

It will thus be seen that the energy requirements of children at different ages may be stated in terms of the requirements of an adult man doing average work. Atwater formulates this as follows:—

Taking a man at . . . 1·0
A woman is equivalent to 0·8 of a man.
A boy of 14 to 16 ,, 0·8 ,,
A girl ,, ,, 0·7 ,,
A child 10 to 13 ,, 0·6 ,,
,, 6 to 9 ,, 0·5 ,,
,, 2 to 5 ,, 0·4 ,,
,, under 2 ,, 0·3 ,,

Mode of Life.—A person kept warm and at rest requires much less energy than if he is required to do muscular work and is exposed to cold.

The energy requirements of the body under different conditions may be determined by measuring the amount of energy given off.

This has been done by enclosing the indivi-

dual to be studied in a Respiratory Calorimeter Chamber.

The most complete and elaborate chamber of this kind which has yet been constructed is that made by Drs. Atwater and Rosa, working under the Board of Agriculture of the U.S.A. Government.

This consists of a double-walled room built in the laboratory, in which a person may be kept for several days at a time. It is provided (1) with an arrangement by which air is supplied and the amount of air measured; (2) with an arrangement by which the heating of the air and the amount of water vaporised may be measured; (3) with an air-tight double window through which the food may be supplied and the excreta passed out. Inside is a folding bed, writing-table and chair, and an ergometer consisting of a fixed bicycle working against a known resistance. With this chamber it is possible to measure—

1. The work done on the bicycle.
2. The heat given off. This is measured by the extent to which water circulating in special coils is heated and water vaporised.
3. The excretion of matter. This is determined by analysing the air entering and the air leaving the chamber, and thus finding the amount of CO_2 and H_2O given off and by analysing the other excretions for nitrogen and carbon.
4. The composition and energy value of the food.

Thus an accurate measurement can be made of the intake of matter and energy in the food and the output of matter and energy from the body, and thus the relationship between them can be determined.

Using this chamber, Atwater and Benedict have made nineteen studies upon three different men who were kept in the chamber for from three to four days, sometimes at rest, sometimes doing mechanical work upon the stationary bicycle.

The general result of these experiments as regards the output of energy is shown in the table.

	Number of Days.	Average Output in Calories.		
		Heat.	Work done.	Total Energy.
Rest .	45	2250	0	2250
Work .	22	3420	234	3654

This shows that a man at rest liberated daily about 2250 calories of energy, while, when doing a very moderate amount of muscular work, he liberates about 3654 calories.

An extensive study of the diets of peoples of all nationalities and of different occupations shows that, unless when poverty prevents it, the food taken per day is capable of yielding in the case of those doing moderate muscular work about 3300 calories, of those at rest about 2700 calories, and of those doing active muscular work 5500 calories.

Benedict states the energy requirement of men doing different amounts of work as follows:—

Sedentary life . .	2700 calories.
Light muscular work .	3050 ,,
Moderate ,, ,, .	3400 ,,
Hard ,, ,, .	4150 ,,

Just as the coal consumption of an engine increases as the work to be done is increased, so the food consumption of man increases with the work to be undertaken.

It might be objected to this method of arriving at a conclusion as to the energy requirements of the body, that the amount of energy eliminated as heat depends simply upon the amount of food taken, and that these results do not indicate what is actually required, but that the surplus food must be got rid of in some way since our power of storing it as fat is fortunately limited; that the body simply behaves like an engine in which, if we put more coal on the furnace, more heat will be given off whether the engine has to work or not. But it has been ascertained that, when the supply of energy in the food is less than the amount which these methods lead us to believe is necessary, the tissues of the body begin to be used, and the body loses weight.

This is very clearly shown by the following observation on convicts. When the observation was begun their diet yielded above 3900 calories per diem, and the waste of food was very great. To obviate this, the diet was cut down to what was considered sufficient to yield the energy required—to yield about 3500 calories. At once complaints of insufficient feeding became rife, waste stopped, and the convicts generally lost weight. The diet was insufficient, and they were living partly on their tissues. The diet was therefore increased to yield about 3700 calories, and the men no longer lost weight.

It must be remembered that the *gross* energy of the diet must be in excess of the *net* requirements of the body, since all the energy of the food is not available (*see* p. 79).

It is therefore safe to conclude that *for a labouring man the supply of energy must be about* 3500 *calories per day.*

III. Protein Requirements.—Since proteins are essential for the building up of the tissues, a certain quantity must be taken in the food. But since any excess of proteins is simply broken up and the nitrogenous part excreted, it is at least uneconomical, if not prejudicial, to take more protein than is necessary. Many experiments have been made to determine the smallest amount of proteins upon which life

can be maintained, and conflicting results have been arrived at. Various investigators have succeeded in maintaining a nitrogenous equilibrium for short periods on an intake of protein no greater than that metabolised in fasting.

Chittenden has recorded a prolonged series of investigations upon this question, by which he shows that in five professional men for periods of from seven to nine months, in eight student athletes for from four to seven months, and in thirteen soldiers for five months, a nitrogenous equilibrium and perfect physical and mental health could be maintained with an intake of from 35 to 56 grms. of protein per diem.

The work done by these individuals is not recorded, but from the daily routine of the soldiers, whose chief work appears to have been two hours' exercise in a gymnasium, it must be classed as very moderate. The fact that the energy value of the diets in the case of the soldiers was only 2500 to 2800 calories seems to confirm the opinion that they were not called upon to do severe work.

While these results certainly prove that men can maintain health and muscular efficiency for long periods on about half the amount of protein which is usually consumed, they do not demonstrate that, in the case of those subjected to strenuous and sustained muscular work, such a reduction is desirable, nor do they indicate that in growing children a reduction in the amount of protein usually consumed may be safely allowed. A study of a very large series of dietaries of different races shows that, unless absolutely prevented by poverty, the average man consumes over 100 grms. of protein per diem, and that those in muscular training tend to consume very much larger amounts. Bearing in mind the importance of proteins as muscle builders, it is safe to conclude that in the labouring classes *about* 120 *grms. of proteins should be allowed per man per diem.*

IV. Part played by Carbohydrates and Fats.—The carbohydrates and fats have to supply the energy not supplied by the proteins. The amounts which must be consumed will thus depend, first, on the amount of protein taken, and, second, on the energy requirement of the individual. If 120 grms. of protein are taken, this will yield 492 calories of energy, while if only 50 are consumed, 205 calories will be yielded. The difference between these amounts and the total energy requirements of the person must be made up from carbohydrates and fats. In the case of a working man requiring 3500 calories, and consuming 120 grms. of protein per diem, this leaves about 3000 calories to be supplied by fats and carbohydrates.

In determining the proportionate amounts of these, two factors have to be considered—first, the relative cost, and, second, the limitation of the power of digestion of each.

Carbohydrates are enormously cheaper than fats as a source of energy. Margarine at 8d. per lb. will yield 435 calories for a penny, while sugar at 2½d. per lb. will yield 1860 calories for the same sum. But the use of carbohydrates as a source of energy is limited by the fact that in most individuals digestive disturbances are apt to supervene if more than from 500 to 600 grms. are consumed.

A diet should therefore not contain much more than 500 grms. of carbohydrates, and these will yield 2050 calories of energy.

This leaves about 950 calories to be supplied by fats, an amount which is nearly met by 100 grms. of fat yielding 930 calories of energy. This quantity of fat most people can easily digest. A typical diet for an average man doing moderate muscular work would thus be—

	Amount.	Energy Value in Calories.
Proteins . .	120	492
Carbohydrates .	500	2050
Fats . . .	100	930
		3472

or very nearly the 3500 calories.

A reference to Atwater's table gives the dietary requirements of a woman or of a child of any age, and thus renders it easy to make out the diet of a family or public institution.

The subject of dietetics cannot be left without alluding to the influence of two very universally used materials—alcohol and tea.

Alcohol.—The influence of alcohol may be considered under two heads.

1*st. Before Absorption.*—Moderate doses of alcohol taken along with food do not appear to interfere with the digestion and absorption of proteins, of carbohydrates, or of fats. When taken apart from food and in such excessive doses as to act as an irritant and to cause catarrh of the alimentary canal, alcohol of course has a prejudicial influence.

2*nd. After Absorption.*—The tissues have the power of oxidising a small quantity of alcohol, and since the combustion of 1 grm. of alcohol liberates 7·06 calories of energy, alcohol must be regarded as a food just in the same way as sugar is a food. The amount of alcohol which can be thus oxidised varies in different individuals, but the average man cannot use more than 50 grms. per diem. This is iso-dynamic with 85 grms. of proteins or carbohydrates. Any alcohol taken above the amount which can be oxidised is excreted in the urine and breath, and in its passage through the body it acts as a poison to the protoplasm, first diminishing its activity and so diminishing the rate of waste, and, secondly, causing the death of the protoplasm and the removal of the broken-down nitrogenous constituents in the urine. Its poisonous action on the liver is demonstrated by the diminished building up of urea, so that a greater quantity of waste nitrogen is excreted

in other forms. In moderate doses it stimulates the heart and dilates the arterioles, and thus no marked change in the blood pressure is produced. On the nervous system its action seems to be purely toxic. Its first action in checking the activity of metabolism, along with its influence in producing dilatation of the cutaneous vessels, is taken advantage of in its use in the treatment of high temperature in some fevers.

Tea, coffee, and cocoa have as their essential constituents the substances theine and caffeine, which are methyl derivatives of xanthin, a diureide which is excreted in the urine (*see* p. 87). They tend to paralyse the sensory mechanism of the brain, and hence abolish the *sense* of fatigue. They stimulate the heart and the secreting action of the kidneys, but they have no effect upon the metabolism. Cocoa nibs contain about 8 per cent of protein and about 50 per cent of fat. Cocoa is therefore an energy-yielding food.

Section VII.—Internal Secretions or Hormones—Their Production and Action

The products of the metabolism of the various organs are carried away in the lymph and blood, and certain of these products exercise an important influence upon other structures in the body. Thus, the carbon dioxide produced in muscle acts upon and stimulates the respiratory centre, and the secretion formed in the lining membrane of the duodenum stimulates the pancreas. These products have been termed internal secretions, while more recently the name Hormones or "Activators" has been suggested for them.

Among the structures which are known to yield such hormones are:—

1. *Suprarenal Bodies.*—These structures lie just above the kidneys. Each consists of a tough *cortex* composed of epithelial-like cells arranged in columns, and a soft *medulla* consisting of cells derived from neuron cells which stain of a peculiar brown colour with chromic acid. The medulla is developed from the sympathetic chain of ganglia. The cortex is a perfectly independent structure derived from the surrounding mesoblast, and in teleostean fishes it is quite apart from the representative of the medullary part. A suprarenal body is thus two distinct and independent organs combined with one another (Fig. 169).

Long ago Brown-Séquard found that removal of these bodies causes great muscular weakness, loss of tone of the vascular system, loss of appetite, and finally death in a short time. Addison had already pointed out that a similar set of symptoms, accompanied by pigmentation of the skin, is associated with diseased conditions of these organs in man. The injection of small quantities of the medullary portion of the bodies, or of extracts of it, exercises a powerful effect on the endings of the thoracico-abdominal visceral nerves. The most marked result is to cause contraction of the arterioles and an enormous rise in the blood pressure. That the action is upon the endings of these nerves is shown by the fact that M'Fie found no action on the heart of the chick in which the nerves are not yet developed, while Brodie has failed to get any

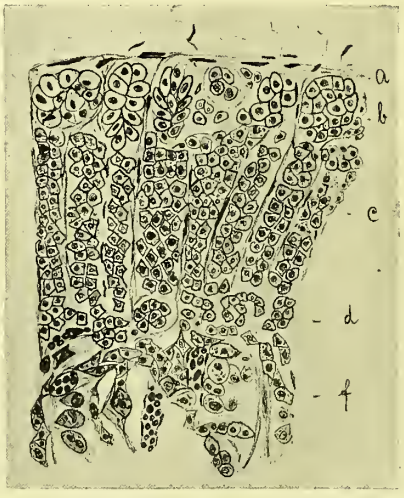


Fig. 169.—Section through cortex and medulla of the suprarenal body of a mammal; *a*, *b*, *c*, *d*, cortex; *f*, medulla.

effect on the vessels of the lungs in which the sympathetic nerve terminations are said to be absent. Further, apocodeine, which poisons the nerve endings, abolishes the effect of suprarenal extracts.

Cardiac inhibition is produced—but this may be due to the rise of blood pressure. It is abolished by the administration of atropin.

These extracts also cause a dilatation of the pupil (vol. vii. p. 537) in the cat, and they inhibit intestinal peristalsis. Their action on the bladder varies according to the action of the sympathetic fibre, which is different in different animals. In those in which these fibres cause contraction, extract of the suprarenal acts in this way; in those in which relaxation is caused, the extract causes relaxation. With blood-vessels which are not supplied with vaso-constrictor fibres, *e.g.* those of the lungs and heart, the extract does not act, and hence a rush of blood to the lungs is caused.

As already indicated (p. 75), injections of extracts of the suprarenal bodies profoundly modify the metabolism, leading to an increase of sugar in the blood and to its excretion in the urine. This is best marked when the animal is well fed and has a store of glycogen in its liver; but since it occurs in fasting animals, after the stored carbohydrates have been cleared out by the administration of phloridzin (p. 74), it would appear to be due, in part at least, either to a non-utilisation of sugar by the tissues or to an increased production of sugar from proteins. A decrease in the nitrogen in the form of urea

and an increase of that in ammonia, similar to that found in cases of true diabetes, have also been observed. It has been suggested that the suprarenal secretion acts through the pancreas by preventing the formation of the internal secretion which has been supposed to act on the liver (see p. 84). But the evidence is not satisfactory.

Its continued administration produces atheroma of the aorta.

The essential principle of the suprarenals is a substance, *adrenalin*, which is of the nature of a secondary alcohol linked to a benzene ring. Various more or less successful attempts have been made to prepare it synthetically.

The functions of the cortex of the suprarenals are unknown.

2. *Pituitary Body.*—This lies at the base of the mid-brain, and consists of an anterior part of nervous tissue, somewhat resembling the medulla of the suprarenals, and a posterior part derived from the alimentary canal, and consisting of masses of epithelial-like cells.

Removal of this body causes in cats and dogs a fall of temperature, lassitude, muscular twitchings, dyspnœa, and ultimately death. Injection of extracts of the substance is said to diminish these symptoms. In the healthy animal the injection of extracts of the posterior or nerve parts of the pituitary causes a rise of arterial blood pressure, a dilatation of the kidney, and an increased flow of urine. A repetition of the injection may cause a fall of blood pressure, but the dilatation of the kidney and the increased flow of urine are produced.

3. *Thyroid Gland* (Fig. 170).—This structure is formed as a hollow outgrowth for the anterior part of the alimentary canal, which branches and again branches. It early loses its connection with the alimentary canal, and becomes cut up by fibrous tissue into a number of small more or less rounded cysts or follicles, each lined with epithelium, and filled with a mucus-like substance, which contains a nucleoprotein, and a substance with a marked power of combining with iodine. This has been called iodothyrin. It contains about 3·6 per cent of iodine, and it seems to be the active constituent of the internal secretion of the gland.

The removal of the thyroid usually leads to a train of symptoms which varies somewhat in different animals, but is essentially the same in nearly all. The connective tissues tend to revert to the embryonic conditions, and the amount of mucin increases. The temperature falls, muscular tremors appear, and in dogs these may go on to convulsions. They do not disappear on removing the cortex cerebri, but are stopped by section of the nerves to the muscles, and thus they appear to be spinal in origin. The functions of the higher nervous system become sluggish, and the animal frequently dies. By administering the substance of the thyroid, or by giving extracts of the thyroid, most of these symptoms may be delayed or prevented. When thyroid gland or extract is given to healthy animals in moderate doses it causes an increased metabolism of both fats and proteins, and may thus induce emaciation. These phenomena seem to indicate that one function of the organ is to produce an internal secretion, which regulates the rate of the metabolic processes in the body by increasing them when such an increase is desirable. When the thyroid is not developed, the growth and development of the individual are partially arrested, and the condition of *cretinism* is produced. Atrophy of the structure in adult life causes a train of symptoms somewhat resembling those produced by its removal, and constituting the disease *myxœdema*. It has been suggested that a condition of increased activity of the action of the heart, usually accompanied by prominence of the eyes and swelling in the region of the thyroid—*exophthalmic goitre*—may be due either to increased activity of the structure or to deficient action of the parathyroids.

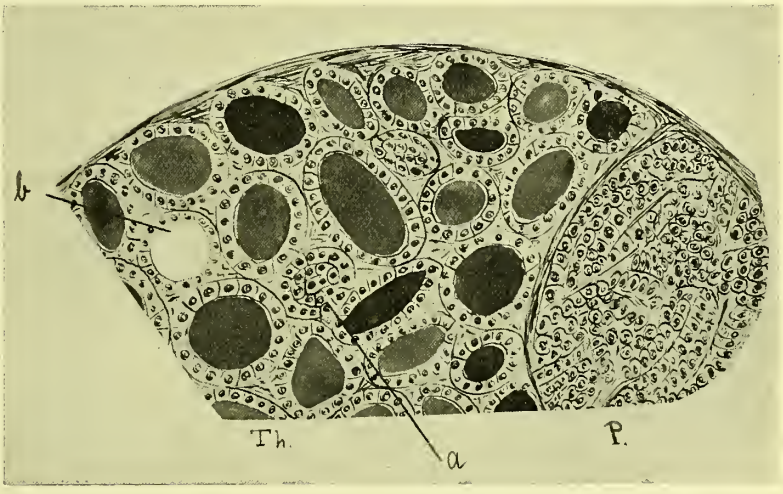


FIG. 170.—Section through part of the thyroid (*Th.*) and a parathyroid (*P.*) of a mammal.

4. *Parathyroids.*—Two to four small nodules are found in close relationship to each lobe of the thyroid, often lying in its substance, and these are formed of columns of cells with capillary blood-vessels between them (Fig. 170). More or less successful attempts have been made in different animals to remove them with-

out the thyroid, or the thyroid without them, and the general result of these experiments is that the nervous symptoms which follow ordinary thyroidectomy—the tremors, etc.—seem to be due to the loss of the parathyroids, while the metabolic changes are probably due to want of the internal secretion of the thyroid. After removal of the thyroid some observers have found that the parathyroids undergo a change and become like the thyroid.

5. *Ovaries and Testes.*—It is well known that removal of these organs causes characteristic changes in the animal: a tendency to the deposition of fat being produced, the activity of the central nervous system being somewhat modified, the voice in the male losing its masculine character, and the thymus persisting, in the male at least, for a considerably longer period than in the normal. That it is not the tubules of the testes but some product of the interstitial cells which acts in this way is demonstrated by the fact that ligature of the vasa deferentia which causes atrophy of the tubules does not prevent the development of sexual characters. Several years ago Brown-Séquard maintained that the general effects of atrophy of these organs might be obviated by the administration of testicular substance; and more recently, as a result of clinical experience, the administration of extracts of the ovaries has been described as relieving certain of the nervous symptoms which supervene on their removal or atrophy. It has further been found that ovarian substance, when given to dogs whether male or female, causes an increase in the rate of protein metabolism, although no similar action is found with testicular substance. There is thus evidence that the ovaries, like the thyroid, form an internal secretion having an important action in accelerating the metabolism. It is at least probable that the testes produce a similar substance.

6. *Thymus.*—This structure develops as an epithelial outgrowth from one or more of the brachial arches of the embryo. Round these outgrowths masses of lymph-like tissue collect, and thus a much-lobulated structure lying in the front of the neck and upper part of the thorax is formed. As development goes on the epithelial core of each lobule breaks up and forms nests of cells which are often disposed concentrically and form the corpuscles of Hassall. These lie in the loose lymph-like tissue, the medulla of the lobule, the meshes of which are formed from the epithelial cells, and this is surrounded by a cortex of more dense lymph-like tissue. The thymus is largest in relationship to the body weight about the time of birth, but it continues to grow, although not in proportion to the growth of the body, till about the age of puberty. After about twenty-four years of age it atrophies and is replaced by a mass of fatty tissue. The Hassall's corpuscles seem to atrophy earlier than the lymph-like tissue.

Castration in cattle and guinea-pigs markedly retards the onset of atrophy, so that the thymus of the ox is much larger than that of the bull of the same age. Not only so, but removal of



FIG. 171.—Section of the lobules of the thymus to show the lobules, with Hassall's corpuscles in the central part.

the thymus in young guinea-pigs seems to be followed by a more rapid growth of the testes. It is therefore probable that the thymus yields an internal secretion which controls the growth of the male genital organs.

The only other effect of its removal in young guinea-pigs is a transient diminution in the number of leucocytes. This seems to lead to a diminished power of resisting the invasion of those micro-organisms—*e.g.* staphylococci—which are normally combated by the leucocytes.

7. *Pancreas.*—Excision of the pancreas in dogs and other mammals produces a condition of diabetes—an increase of sugar in the blood, its appearance in the urine, an increased excretion of nitrogen, and a general emaciation. These symptoms do not occur when the duct is tied or occluded until degeneration has developed, but they are invariable and immediate when a sufficient amount of the gland is removed. They are not prevented by the administration of pancreas, either fresh or as extracts. The sugar is formed from the proteins, since it appears in amounts proportionate to the amount of nitrogen excreted, after all the glycogen has been removed. The pancreas seems therefore to form a hormone which either controls the production of sugar in the liver or causes its utilisation by the muscles.

It has been suggested that adrenalin causes diabetes by checking the production of this hormone in the pancreas. But the facts that, in ducks and geese, in which removal of the pancreas does not cause diabetes, the injection of suprarenal extracts causes glycosuria, and that its administration increases the output of sugar in dogs without a pancreas, are opposed to the view that it acts through the pancreas,

and suggest that it must act directly on the liver or other tissues.

Since the only respect in which the pancreas differs in histological character from the parotid gland—removal of which has no effect on the metabolism—is in the presence of the islets of Langerhans, it has been suggested that they are related to this function of the organ. Some recent observations, however, tend to show that these islets are not permanent structures, but that they are formed from and revert to the ordinary secreting tissue.

8. *Duodenum.* — As already pointed out (p. 66), the duodenum yields a hormone (*secretin*) which acts directly upon the pancreas to stimulate its secretion.

Toxic Action and Immunity

It is not definitely known how each of these internal secretions performs its special action, but light seems to be thrown upon the question by the study of the mode of action of various toxic substances, and the mode of production of a condition of immunity against them. As will be presently shown, a process of the same nature as the production of internal secretions is involved.

Snake and Diphtheria Toxins.—The question may be most simply approached by considering first the probable mode of action of the toxin or poison of snake venom, or of that produced by the diphtheria bacillus, and the way in which protection against these is established by the development of antitoxins.

By injecting under the skin of the horse increasing doses of such toxins the animal becomes quite resistant to the poison. A certain quantity of its serum can then neutralise a definite quantity of the toxin, so that if the mixture of serum and toxin be injected the animal is uninjured. Something has been formed in the horse which seizes on the molecules of the toxin and makes them harmless, just as when soda is added to sulphuric acid it forms a neutral salt.

The two molecules have a definite chemical affinity for one another, so that the toxin is no longer free to seize upon the protoplasm of the animal's body. To explain this Ehrlich has suggested that the protoplasm molecule, like the protein molecule (vol. vii. p. 490), is to be considered as made up of a central core with a number of side-chains, hands, or *receptors*, which play an important part in taking up nourishment of different kinds, for each variety of which special side-chains have a special affinity (Fig. 172). He supposes that some of these side-chains fit the toxin molecule, and are thus capable of anchoring it to the cell and allowing it to exercise its toxic action, and he explains the production of antitoxin by supposing that, as these side-chains get linked to the toxin and are thus, as it were, thrown out of action, others are produced to take their place, since they are necessary for the nourishment of the protoplasm. If the toxin is continually administered in small doses this production of side-chains may be so increased that they get thrown off into the blood and in it are capable of linking to the toxin and so preventing it from fixing itself to the cells. If, therefore, some of the blood be injected into an animal which afterwards receives a dose of the toxin, that toxin will not act, and the animal will be immune.

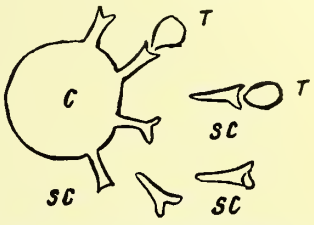


FIG. 172.—To illustrate the formation of side-chains or receptors, *sc*, by which the toxin molecules, *T*, are either anchored to the cell or neutralised. When the side-chains are set free an anti-toxin is formed.

Typhoid Toxin.—But immunity may also be established not against toxins separate from organisms, but against organisms which hold their toxin, as in the case of the bacillus of typhoid fever. Here repeated injections of increasing doses produce a serum which has the power of destroying the organism when added to it even outside the body. But this is not a simple combination, because if the serum be heated to 55° C. it loses its power; but if a few drops of the fresh serum of an unimmunised animal be added, the power is restored. Obviously the anti-body which destroys the organism —the *bactericidal* or *bacterilytic body*, often called the *amboceptor*—requires the co-operation of another body to enable it to act, and this body has been called the *complement* or *activator*. Ehrlich supposes that the immune body does link to the protoplasm of the organism, but that it must in its turn be linked to the complement. The figure may help to explain this (Fig. 173).

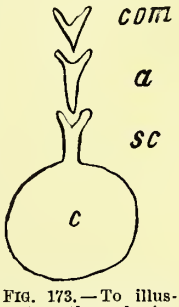


FIG. 173.—To illustrate the anchoring of the anti-body or amboceptor, *a*, to the cell by a side-chain or receptor, *sc*, and the action upon it of complement, *com*.

Cytotoxins. — Similar antibodies, acting upon the cells of the animal body, may be produced by injecting the particular kind of cell into an animal of another species. Thus, if human blood be repeatedly injected into a rabbit the serum of the rabbit's blood becomes *hæmolytic—i.e.* acquires the power of dissolving the erythrocytes in *human* blood. In this case too, the immune body requires the presence of a complement, readily destroyed at a comparatively low temperature, to enable it to act. If such hæmolytic serum be injected into another animal an anti-hæmolysin may be developed—a body which will antagonise the action of the hæmolysin. Possibly this is a body which links

with the amboceptor to prevent its linking to the complement.

Precipitins.—By the injection of the proteins of the blood of any particular animal into an animal of another species a serum is developed which precipitates the proteins of the blood of the first species and of no others.

In these various cases the active body is produced by the throwing off of side-chains from protoplasm, and as these products are carried away in the blood the process is exactly analogous to the formation of internal secretions.

Opsonins. — Many bacteria after treatment with the serum of the animal are taken up by the leucocytes, but if not treated with serum are not taken up. Apparently the serum contains something, which has been called an opsonin, which prepares the bacteria to be devoured. The action of opsonins is destroyed by temperatures between 55° and 65° C. The opsonic power of the serum is often increased by the injection of small quantities of dead bacteria (vaccines).

Section VIII.—Excretion of Matter from the Body

A. Excretion by the Lungs. B. Excretion by the Kidneys

Urine.—The water and waste nitrogen of the body are chiefly eliminated in the urine, which is secreted by the kidneys.

The tests for the various constituents of the urine must be studied practically.

I. *Physical Characters.*—The characters of the urine depend largely on the relative proportion of water and of solids which are excreted in it. At one time it may be very concentrated, while at another time it may be very dilute. For this reason its specific gravity, which depends upon the percentage of solids in solution, varies within wide limits, being often as high as 1030 and frequently as low as 1005; but the average specific gravity is about 1020. It is possible from the specific gravity to form a rough idea of the amount of solids present, for, by multiplying the last two figures by 2·22 the amount of solids per 1000 parts is given approximately.

Since the percentage of pigments in the urine varies like that of the other constituents, the colour of the urine shows wide divergence in the normal condition. A concentrated urine has a dark amber colour, while a dilute urine may be almost colourless. Under average conditions the urine has a straw yellow colour.

The reaction of urine is normally acid in man, chiefly from the presence of acid sodium phosphate, NaH_2PO_4, and the degree of acidity varies with the concentration. But the acidity of the urine may also be varied by different conditions. It is increased when there is an increased oxidation of proteins, for the sulphuric acid and phosphoric acid thus formed have to be neutralised by combining with the alkalies. It is decreased by taking alkalies, or by taking such vegetable salts as the citrate, malate, or tartrate of sodium or potassium, because these are oxidised in the body and excreted as the carbonates. The NaH_2PO_4 may then be changed to Na_2HPO_4.

Urine is normally transparent; but when it has stood for a few hours, a transparent cloud is seen floating in it. This consists of a mucin-like substance of the nature of a nucleo-protein. When urine is alkaline it is turbid from the separation of a white deposit of calcic and magnesic phosphates. In the alkaline urine of herbivora the white deposit is chiefly composed of calcium carbonate.

A brick-red deposit of urates tends to fall as the urine cools when it is concentrated and very acid.

The smell of urine is characteristic, and it may be modified by the ingestion of many different substances.

II. *Composition.*—Since the relative amounts of water and solids vary within such wide limits, the percentage composition of urine is of little moment. Under average conditions the water constitutes about 96 per cent, and the solids about 4 per cent. Of these solids, rather more than half are organic, rather less than half are inorganic. Since water and solids are derived from the water and solids taken by the individual, the amounts excreted depend upon the amounts taken, and must be considered in connection with them. Thus, if a man takes little fluid, he will pass little water in the urine. If he takes little food, a small quantity of solids will be excreted by the kidneys.

Since excretion and ingestion must be studied in relationship to one another, it is convenient to compare them during a definite period of time, and the natural division into days of twenty-four hours is generally adopted.

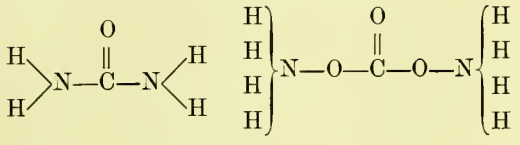
Under ordinary conditions the amount of solid food taken per day does not vary very greatly, but the amount of fluids imbibed varies within much wider limits. For this reason, while the amount of water excreted in the urine per diem varies enormously, the amount of solids is more fixed.

In a man on an average diet about 1500 c.c. of water and 60 to 70 grms. of solids are daily eliminated.

I. Nitrogenous Substances. — A. *Urea.*— Urea is the most important constituent of the urine. Its chemistry and mode of formation have been discussed on page 75. Since it is as urea that, on an ordinary diet, nearly 90 per cent of the waste nitrogen is eliminated in the urine, the amount excreted depends upon the amount of proteins taken in the food. For this reason, during fasting, the excretion of urea may fall as low as 6 grms. per diem, while on a diet containing the ordinary amount of proteins, about 33 grms. of urea—15·4 grms.

of nitrogen—are excreted. On a normal diet from 86 to 90 per cent of the waste nitrogen is excreted as urea; but, when the protein intake is decreased, carbohydrates and fats being substituted, the proportion of urea-nitrogen may fall to as low as 60 per cent.

When the urine is allowed to stand, certain micro-organisms are apt to get into it, and to cause a hydration of the urea, whereby it is changed into ammonium carbonate—

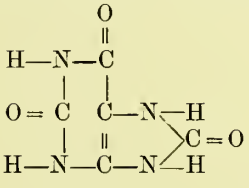


The urine is thus made alkaline, and the phosphates of the earths are precipitated. The magnesium phosphate combines with the ammonia to form ammonio-magnesium-phosphate, $NH_4MgPO_4 + 6H_2O$, which crystallises in characteristic prism-like crystals.

B. *Non-Urea Nitrogen.*—The 10 or 12 per cent of nitrogen which, on an ordinary diet, is not excreted as urea is distributed in :—

1. *Ammonium Salts.*—About 4 or 5 per cent of the total nitrogen is normally excreted as ammonium salts. But under certain conditions the proportion is greatly increased. Anything which causes an increased breaking down of protein and an increased formation of acids leads to an increased excretion of ammonia—the ammonia being formed from the proteins to neutralise the acids.

2. *Diureides.*—The members of this series of bodies consist of two unmodified or modified urea molecules, linked together by an acid nucleus. The most important have as the linking molecule an oxi-acid with three carbons in series, and they constitute the purin bodies.



In birds and reptiles they largely replace urea as the substances in which nitrogen is chiefly eliminated. In these animals they are formed in the liver from the various products of the decomposition of protein molecules, but in mammals they appear to be very largely derived from the decomposition of nucleic acid. Even when all supplies from without of nucleins and purin bodies are cut off, a certain amount of these purin bodies is daily eliminated. These have been called the "endogenous" purins, while those derived from the constituents of the food are termed the "exogenous" purins. A certain amount of the purins formed are changed to urea before being excreted, and therefore, when disturbances of the chemical processes in the liver occur, the purins may be increased at the expense of the urea.

Uric acid is the most important member of the series. Its constitution is shown above.

It is an exceedingly insoluble substance which tends to crystallise in large irregular crystals, and in the urine these are generally coloured brown by the urinary pigment.

It occurs as salts of sodium and potassium, and, according to Roberts, the acid salt $NaH\bar{U}$ is linked to a molecule of the acid to form $NaH\bar{U}\text{-}H_2\bar{U}$, or what he calls a quadriurate. The evidence of this is not conclusive.

Although the salts of uric acid are more soluble than the free acid, only a small quantity can be dissolved in the urine. Apparently inorganic salts, such as sodium phosphate, act as solvents. When the urine cools, especially if it is unusually acid, the urates tend to separate out and fall as a brick-red deposit, which generally shows no crystalline structure under the microscope.

If the urine has become ammoniacal on standing, the deposit frequently contains characteristic spinous pigmented crystals of ammonium urate.

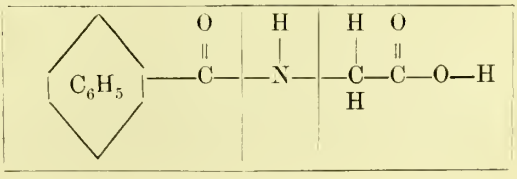
From their insolubility, uric acid and the urates tend to form calculi or concretions in the urinary passages. The presence of uric acid in such concretions is recognised by the *murexide test*, which depends upon the fact that uric acid heated with nitric acid is oxidised to alloxantin, which strikes a purple colour with ammonia, yielding murexide—the ammonium salt of purpuric acid.

Other members of the series, such as xanthin and hypoxanthin, occur in the urine in small quantities.

Allantoin, which occurs in the urine of the fœtus and in the urine of dogs after the administration of nucleic acid, is a diureide in which glyoxylic acid with two carbon atoms is the linking band.

3. *Creatinin.* — Creatinin is formed from creatin (vol. vii. p. 503) by dehydration. The amount excreted is always small, and probably depends upon the amount of muscular tissue broken down in the body. According to the investigations of Folin the amount excreted per diem on a flesh-free diet is very constant in each individual, and does not vary with the amount of protein food taken. Creatin may also appear in the urine in small amounts.

4. *Hippuric Acid.*—This is benz-amino-acetic acid—



It is formed from benzoic acid taken in the food by linking it to glycocoll—amino-acetic acid. This synthesis appears to take place in the kidneys, for it has been found that hippuric acid is not formed when these organs are excised, and that, when blood containing benzoates is circulated through them, hippuric acid is produced. Its chief interest is in the fact that it is one of the first organic compounds which were demonstrated to be formed synthetically in the animal body. Normally it is present in human urine in very small quantities, but in the urine of herbivora the amount is considerable, from the presence of benzoic acid in the fodder. The acid itself is insoluble, and it occurs as the soluble sodium salt.

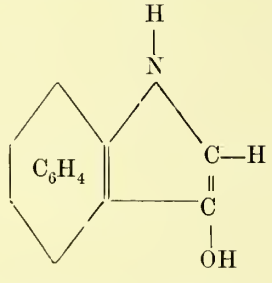
II. Sulphur-Containing Bodies.—The sulphur excreted in the urine is derived from the sulphur of the protein molecule, and the amount of sulphur excreted may be taken as a measure of the amount of protein decomposed. This is sometimes used as a check upon the estimation from the excretion of nitrogen.

A. *Acid Sulphur.*—The greater part of the sulphur is fully oxidised to SO_3.

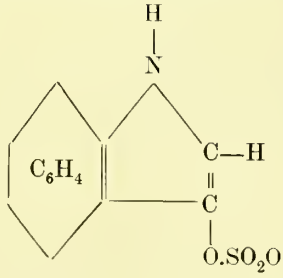
(*a*) *Preformed Sulphates.*—About nine-tenths of this is linked with bases to form ordinary sulphates.

(*b*) *Ethereal Sulphates.*—The other one-tenth is in organic combination, linked to benzene compounds formed by oxidation of the indol, skatol, and phenol (*see* p. 69) which are produced by the putrefaction of proteins in the bowel.

Indol is oxidised into indoxyl thus—



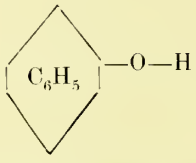
This when linked to sulphate of potassium forms indoxyl-sulphate of potassium or indican.



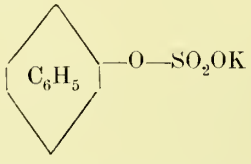
From skatol, which is methyl-indol, skatoxyl-sulphate of potassium is formed in the same way.

These bodies are colourless, but when oxidised they yield pigments—indican yielding indigo blue, skatoxyl-sulphate of potassium yielding a rose colour.

Phenol—

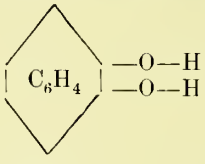


is also linked with potassium sulphate, and excreted in the urine.



The amount of these ethereal sulphates depends upon the activity of putrefaction in the intestine, and is a good index of its extent.

When dioxybenzene or pyrocatechin—



is formed in the body, it too is linked to sulphate of potassium and excreted. When urine containing this substance stands, it becomes oxidised and yields a greenish brown or black pigment.

B. *Neutral Sulphur.*—A small quantity of sulphur is sometimes excreted in a less oxidised state, in the form of *neutral sulphur*. The most important compound of this kind is *cystin*, the disulphide of amino-propionic acid—two molecules of amino-propionic acid linked by sulphur—

Amino-propionic acid.
|
Sulphur.
|
Sulphur.
|
Amino-propionic acid.

In some individuals, and in certain conditions of the metabolism not yet fully understood, the amount of cystin is increased, and it then tends to crystallise out of the urine in peculiar hexagonal plates.

III. Phosphorus-Containing Bodies.—The phosphorus in the urine is derived partly from phosphates taken in the food, and partly from the nucleins of the food and tissues and from the bones.

(*a*) Normally the phosphorus is fully oxidised to P_2O_5, which is linked to alkalies and earths

and excreted in the urine. The most important phosphate is the di-hydrogen sodium phosphate NaH_2PO_4, which is the chief factor in causing the acidity of the urine. About *one-quarter* of the phosphoric acid is linked to *calcium* and *magnesium*, and it is these earthy phosphates which precipitate when the urine becomes alkaline. When the urine becomes ammoniacal, triple phosphate is formed (p. 87).

(*b*) It is probable that a small quantity of the phosphorus is excreted in organic compounds, such as glycero-phosphates; but so far these have not been fully investigated.

IV. Chlorine-Containing Bodies.—Sodium chloride is the chief salt of the urine. It is entirely derived from the salt taken in the food, and its amount varies with the amount ingested. From 10 to 15 grms. are usually excreted per diem in a person on a normal diet.

In starvation to a certain extent, and very markedly in fever, the tissues of the body have a great power of holding on to the chlorine, and the chlorides may almost disappear from the urine.

V. Bases of the Urine.—Sodium, potassium, calcium, and magnesium occur in the urine in amounts varying with the amounts taken in the food. Generally speaking, sodium is in excess of the others, but on a flesh diet and in starvation it falls below the potassium. Calcium and magnesium are present in much smaller quantities.

VI. Pigments.—A brown hygroscopic substance, which gives no bands in the spectrum, may be extracted from urine. This has been termed urochrome. By reducing this, another pigment, urobilin, is produced, which gives definite bands, and which is frequently present in the urine. It is probably identical with the hydrobilirubin which has been prepared from the bile pigments, and it contains C, H, O, and N.

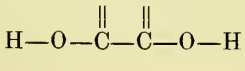
The pigment which gives the pink colour to urates has been called uroerythrin, and its chemical nature is unknown.

Hæmatoporphyrin (*see* p. 6) is normally present in small traces in the urine, but in certain pathological states it is increased in amount and gives a brown colour to the urine.

VII. Nucleo-protein.—A mucin-like nucleo-protein derived from the urinary passages is always present in small amounts, and forms a cloud when the urine stands.

VIII. Carbonic and Oxalic Acids.—1. *Carbonic Acid.*—Small amounts of this are present in urine, and after the administration of citrates, malates, or tartrates, the amount may be considerably increased, and the urine may then effervesce strongly when an acid is added.

2. *Oxalic Acid*



is a substance in a stage of oxidation just above that of carbonic acid. It is frequently present in the urine linked with calcium, and the calcium salt tends to crystallise out in characteristic octohedra, looking under the microscope like small square envelopes. Under certain conditions these crystals assume other shapes. The oxalic acid of the urine is chiefly derived from oxalates in vegetable foods, but it has been detected in the urine of animals on a purely flesh diet.

Secretion of Urine.—*Structure of the Kidney.*—The kidney (Fig. 174) is a compound tubular gland, consisting of innumerable tubules, each made up of—

(1) A closed extremity or Malpighian body (*M.B.*), consisting of an expansion at the end of the tubule—Bowman's capsule—into which a tuft of capillary vessels—the glomerulus—projects.

(2) Extending away from this is a *proximal convoluted tubule* (*P.C.T.*) lined by pyramidal

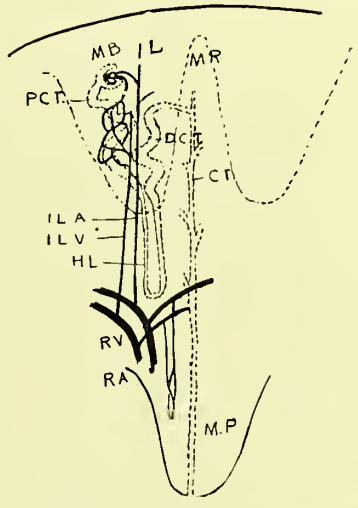


Fig. 174.—Diagram of the structure of the kidney. *M.P.*, Malpighian pyramid of the medulla; *M.R.*, medullary ray extending into cortex; *L.*, labyrinth of cortex; *M.B.*, a Malpighian body consisting of the glomerular tuft and Bowman's capsule; *P.C.T.*, a proximal convoluted tubule; *H.L.*, Henle's loop on the tubule; *D.C.T.*, distal convoluted tubule; *C.T.*, collecting tubule; *R.A.*, branch of renal artery giving off *I.L.A.*, interlobular artery, to supply the glomeruli and the convoluted tubules; *I.L.V.*, interlobular vein bringing blood back from the cortex.

and granular epithelial cells. This dives into the medulla, becomes constricted and lined by a transparent flattened epithelium, and is known as the *descending limb* of the looped tubule of Henle. Turning suddenly upwards and becoming lined by a cubical granular epithelium, it forms the *ascending limb*, and reaching the cortex expands into the *distal convoluted tubule*, which exactly resembles the proximal (*D.C.T.*). It opens into a *collecting tubule* (*C.T.*) lined by a low transparent epithelium, which conducts the urine to the pelvis of the kidney.

The renal artery breaks up and gives off a series of straight branches—the interlobular

arteries (*IL.A.*)—which, as they run towards the surface, give off short side branches which terminate in the glomeruli. The efferent vein passing from these breaks up again into a series of capillaries between the convoluted tubules, and these pour their blood into the interlobular veins (*IL.A.*). This arrangement helps to maintain a high pressure in the capillary loops of the glomerular tuft.

Physiology of Secretion.—The marked difference in the structure and vascular supply of the Malpighian bodies and of the different parts of the renal tubules makes it probable that they perform different functions.

Malpighian Bodies.—(*a*) It has been shown by injecting into the blood-vessels acid fuchsin, which is colourless in alkaline solution and red in acid solution, that the urine formed in these bodies is alkaline in reaction and that it becomes acid as it passes down the convoluted tubules.

(*b*) It is also known that these bodies are thrown out of action by decreasing the flow of blood through the kidneys. The rate of flow of blood depends upon the blood pressure in the renal arteries and the dilatation of the renal arterioles. It may be measured by measuring the amount of blood passing out by the renal vein, or by enclosing the kidney in an oncometer —a closed vessel connected with a recording tambour—so that changes in the volume of the kidney are recorded (*see* p. 20), or by a combination of these methods.

Section of the renal nerves derived from the 11th, 12th, and 13th dorsal nerves causes a dilatation of the renal arterioles, an expansion of the kidney, and an increased flow of urine. Stimulation of these nerves has the opposite effect. A fall in the general arterial pressure to about 50 mm. Hg in the dog generally causes a decreased flow of blood through the kidney and practically stops the flow of urine, although the tubules, as will be presently shown, still act.

(*c*) In the frog the renal arteries supply the Malpighian bodies, while portal veins from the posterior end of the animal supply the convoluted tubules. Ligature of the renal arteries stops the flow of urine, but the flow may be again induced when urea is injected.

(*d*) Even when this flow is induced, if dextrose or egg albumin or peptone are injected into the blood, substances which in the normal frog appear in the urine, they are not excreted.

These observations seem to show that the Malpighian bodies have to do chiefly with the excretion of water and of certain abnormal constituents of the blood, and that their activity depends upon the rate of blood-flow through them. That it is not due to a mere physical filtration under pressure is indicated by experiments, in which the blood pressure in the kidneys of a dog was raised by injecting large quantities of blood from another dog, without the flow of urine being increased. That it is a selective action of the epithelium seems to be proved by the passage into the urine of such large molecules as those of egg albumin and hæmoglobin and of various pigments such as carmine.

The fact that, if a cannula connected with a manometer be placed in the ureter, the secretion of urine stops when a pressure of about 50 mm. Hg is reached, is not opposed to the view that the formation of urine from the glomeruli is an active secretion. In the salivary gland, where the formation of saliva is undoubtedly due to the vital action of the cells, the same result follows obstruction of the duct.

The point of practical importance is that *the secretion of water takes place chiefly through the Malpighian bodies*, and that this is reduced or stopped by a fall in the general arterial pressure, such as occurs in heart failure. The decreased excretion of water may lead to the development of dropsy.

The Tubules.—The alkaline urine formed in the Malpighian bodies undergoes changes as it passes along the tubules. It becomes acid and the various solids are increased, for urine contains a higher proportion of these than does the blood, *e.g.* the blood contains only about 0·03 per cent of urea, but the urine usually contains 2 per cent. The addition of solids in the tubules is further proved by the following facts :—

(*a*) Uric acid crystals are frequently found in the cells of the convoluted tubules of the kidney of birds. (*b*) Heidenhain, by injecting a blue pigment—sulph-indigotate of soda—into the circulation of the rabbit, demonstrated that the cells of the convoluted tubules take it up and pass it into the urine. In the normal rabbit the whole of the kidney and the urine become blue. But, if the formation of urine in the Malpighian bodies be stopped by cutting the spinal cord in the neck so as to lower the blood pressure, then the blue pigment is found in the cells of the convoluted tubules and of the ascending limb of Henle's tubule. (*c*) When the Malpighian bodies of the frog have been thrown out of action by ligaturing the renal arteries, the injection of urea still causes a flow of urine and the excretion of urea by the tubules. When the portal veins which supply the tubules were ligatured on one side, it was found that less urine was formed on the ligatured than on the unligatured side.

These last experiments show that the cells of the convoluted tubules are capable of secreting water as well as solids, and that they can do so is further demonstrated by the fact that, if by cutting the spinal cord in the neck the formation of urine in the Malpighian bodies of a dog is stopped, the administration of caffeine and of some other substances causes an increased flow

of urine, although the blood pressure in the kidneys is not raised. This is taken advantage of in cases of heart disease, when the secretion of urine is almost arrested from low arterial pressure, and when dropsy is rapidly advancing. Until the heart is toned up, the kidneys may be stimulated to get rid of water by means of such diuretics as caffeine.

It is quite possible that under certain conditions the cells of the renal tubules absorb water and possibly the other constituents of the urine, but the weight of the evidence, we at present possess, is against the idea that they exercise the function to any great extent.

Excretion of Urine. — 1. *Passage from Kidney to Bladder.*—The pressure under which the urine is secreted is sufficient to drive it along the ureters to the bladder. If these are constricted the pressure behind the constriction rises, and may distend the ureters and the pelvis of the kidney; but when it reaches about 50 mm. Hg in the dog, the secretion of urine is stopped. The muscular walls of the ureters show a rhythmic peristaltic contraction, which must also help the onward passage of the urine to the bladder.

2. *Micturition.*—As the urine accumulates in the urinary bladder the rhythmic contraction of the non-striped muscle becomes more and more powerful. These contractions are chiefly excited by the fibres of the nervi erigentes of the second and third sacral nerves, although fibres passing down from the inferior mesenteric ganglion also probably act either in exciting or inhibiting them in certain animals. The backward passage of the urine into the ureters is prevented by the oblique manner in which these tubes pass through the muscular coat of the bladder.

The passage of urine into the urethra is at first prevented either by the oblique manner in which the urethra leaves the bladder, or more probably by the contraction of a strong band of non-striped muscle, the *sphincter trigonalis*. This muscle or the striped fibres which surround the membranous part of the urethra are under the control of a centre in the lumbar enlargement of the spinal cord, and the expulsion of urine must be preceded by their relaxation. In some cases of inflammation of the spinal cord the increased activity of the centre may prevent the expulsion of urine, while later in the disease, when the nerve structures have been destroyed, the urine is not retained and dribbles away on account of the absence of the tonic contraction of the muscles.

The expulsion of the last drops of urine is carried out by the rhythmic contraction of the bulbo-cavernous muscle, while the peristaltic contraction of the bladder wall is assisted by the various muscles which press upon the contents of the abdomen and of the bladder.

In man, in early life, micturition is a purely reflex act, and in the dog it is perfectly performed when the spinal cord is cut in the back. As age advances the reflex mechanism comes to be more under the control of the higher centres, and the activity of the sphincters may be increased or abolished as circumstances indicate.

C. Excretion by the Skin

The skin is really a group of organs, and some of these have been already studied.

(1) The *protective* functions of the horny layer of epidermis, with its development in hair and nail, and of the layer of subcutaneous fat, are manifest.

Hair.—In man the hair has largely ceased to have the important protective function it fulfils in many of the lower animals, but the muscular mechanism by which the position of the hairs can be modified still persists. Attached to each hair follicle is a band of non-striped muscle, the *arrector pili*, which can erect the hair by contracting. These muscles are under the control of the central nervous system, and the nerve fibres have been demonstrated in the cat to take much the same course as the vaso-constrictor fibres of somatic nerves (*see* p. 33). A hair after a time ceases to grow, and the lower part in the follicle is absorbed and the hair is readily detached. From the cells in the upper part of the follicle a new down-growth occurs, a papilla forms, and the hair is regenerated. In many of the lower animals this process occurs twice a year.

(2) The *sensory* functions have been studied under the Special Senses (vol. vii. p. 527).

(3) The *respiratory* action of the skin in mammals is of little importance.

(4) *The Excretory Function of the Skin.*—Three sets of glands develop in the skin—sweat glands and sebaceous glands, which are common to both sexes and are constantly active—and mammary glands, which are active in the female during the period of suckling.

A. Sweat Secretion — 1. *Structure.* — The sweat glands are simple tubular glands coiled up in the subcutaneous tissue with ducts opening on the surface of the skin. The secreting epithelium somewhat resembles that of the convoluted tubules of the kidney. It has been calculated that a man possesses about two and a half million sweat glands, and that if spread out they would present a surface of very great extent.

2. *Functions.*—From these glands a considerable amount of sweat is poured out; but to form any estimate of the daily amount is no easy matter, since it varies so greatly under different conditions. Probably about 1000 c.c. is an average amount. When poured out, sweat usually evaporates, and is then called *insensible perspiration*; but when large quantities are formed, or when, from coldness of the surface, or of the air, or from the large quantity of watery vapour already in the air, evaporation

is prevented, it accumulates, and is called *sensible perspiration.*

A free secretion of sweat is usually accompanied by a dilatation of the blood-vessels of the skin, but this may be absent, and it may occur without any sweat secretion—*e.g.* under the influence of atropine.

3. *Nervous Mechanism of Sweat Secretion.*—The sweat glands are under the control of the central nervous system. This may be very conveniently studied in the cat, in which animal the sweat glands are chiefly in the pads of the feet. If a cat be put in a hot chamber it sweats on the pads of all its feet. But if one sciatic nerve be cut the foot supplied remains dry. If the cat be placed in a warm place and the lower end of the cut sciatic stimulated, a secretion of sweat is produced. These sweat-secreting fibres all pass through the sympathetic ganglia, and back into the spinal nerves. Those to the leg and foot come from the upper lumbar region, those for the hand and arm from the fifth to the eighth cervical nerve, and those for the head in the sympathetic of the neck and partly in the fifth cranial nerve.

The centres presiding over these nerves are distributed down the medulla and cord. They are capable of (*a*) reflex stimulation, as when pepper is taken into the mouth; and (*b*) of direct stimulation by a venous condition of the blood, as in the impaired oxygenation of the blood which so frequently precedes death as the respirations fail.

But even after the nerves to the sweat glands are cut, the glands can be stimulated by certain drugs—*e.g.* pilocarpine. The action of heat seems also to be chiefly peripheral, setting up an unstable condition of the gland cells so that they respond more readily to stimulation.

4. *Chemistry of Sweat.*—Sweat is a clear, watery fluid, with a faintly acid reaction from admixture with sebum and from the presence of acid sodium phosphate. It has a characteristic odour. Its specific gravity is low, about 1004, and it contains less than 2 per cent of solids, of which the chief is NaCl. Of the organic solids, urea is the most important. About 4 or 5 per cent of the total nitrogen excreted from the body is thrown off by the skin in this form, and, when the action of the kidneys is interfered with, very considerable quantities may be eliminated. The sweat nearly always contains epithelial squames from the epidermis, and oil globules derived from the sebum.

B. Sebaceous Secretion. — The sebaceous glands are simple racemose glands which open into the hair follicles, and their function is to supply an oily material to lubricate the hairs. This secretion is produced by the shedding and breaking down of the cells formed in the follicles of the glands. Those lining the basement membrane are in a condition of active division, but the cells thrown off into the lumen of the follicle disintegrate and become converted into a semi-solid oily mass, which consists of free fatty acids and of neutral fats of glycerine and of cholesterin. These cholesterin fats are the *lanolins*, which differ from ordinary fats in being partly soluble in water. Free cholesterin is also present in the sebum.

C. Milk Secretion.—1. *Physiology.*—Before pregnancy occurs the mammary glands are largely composed of fibrous tissue, with a large amount of fat, in which run the branching tubules of the glands as small solid blocks of cells.

As pregnancy advances these tubules grow outwards and increase, and the cells begin to divide, some remaining attached to the basement membrane, some coming to lie in the middle of the tubules. These latter undergo a fatty change and break down, and they are shed in the first milk which is secreted, the colostrum. The cells left upon the basement membrane elaborate the constituents of milk, and the presence of fat globules in their protoplasm is very manifest.

The milk, after being secreted, collects in the ducts of the glands and in the sinus below the nipple, and is expelled from these by the contraction of the muscular fibres in their walls, and by the suction of the young animal. The *excretion* of milk from the ducts is directly under the control of the nervous system, but the evidence as to the way in which the central nervous system influences the *secretion* of milk is by no means satisfactory. Clinical experience shows that it is profoundly modified by nervous changes, but so far stimulation of the nerves to the glands has not yielded definite results.

The chemistry of milk is considered upon p. 54, and the way in which fats and proteins are formed is dealt with on p. 74. The production of the disaccharid lactose and of the phospho-protein caseinogen implies a synthetic process in the protoplasm of the cells of the mammary glands.

PART III.—Reproduction

See Embryology; Fœtus and Ovum, Development of; Generation, Female Organs of; Labour; Menstruation; Pregnancy; Puerperium; Scrotum and Testicle (*Anatomy of the Testis*); etc.

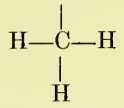
Appendix—Organic Chemistry

The following elementary facts may help the reader who has neglected the study of the outlines of organic chemistry in understanding the chemical problems of physiology.

Organic compounds are built round the four-handed carbon atom

When each hand links to the one-handed hydrogen atom,

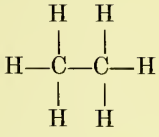
METHANE—



is formed.

By taking away a hydrogen atom from two methane molecules and linking these molecules together

ETHANE—

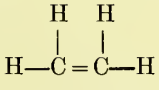


is produced.

By further linking more and more of these molecules together, similar molecules containing three, four, five, or more carbon atoms are produced.

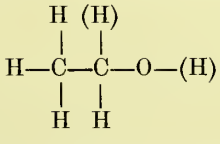
When each carbon has its due proportion of hydrogen atoms it is *saturated*, but if two hydrogen atoms are let go, the unoccupied hands of the carbon may join and form an *unsaturated* molecule, thus :—

Ethane becomes Ethylene



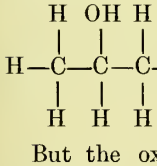
When one hydrogen atom is taken away and the molecule has a hand ready to link with some other substance a *radical* is constituted, and these are known as METHYL, ETHYL, etc.

Alcohols.—When the two-handed oxygen atom O linked to hydrogen H, and thus forming the hydroxyl molecule OH, is linked to the vacant hand of the radical, an alcohol is formed, *e.g.*—



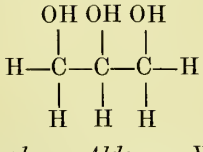
Ethyl Alcohol.

When the terminal carbon is thus oxidised a *Primary Alcohol* is formed; but if a middle carbon atom is oxidised, a *Secondary Alcohol* is produced—



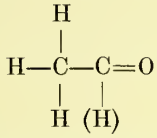
Secondary Propyl Alcohol.

But the oxidation may involve more than one carbon atom, and thus the *Polyvalent Alcohols* are produced—



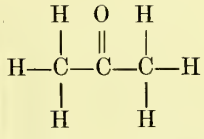
Glycerin.

Aldehydes or Aldoses.—When from a primary alcohol the two hydrogens marked above in brackets are removed, the vacant hand of the oxygen links to the vacant hand of the carbon to form an Aldehyde or Aldose—



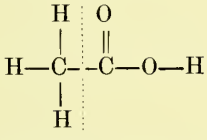
Ethyl Aldehyde.

Ketones or Ketoses.—These are formed in the same way from the Secondary Alcohols, thus :—



Acetone, the Ketose of Secondary Propyl Alcohol.

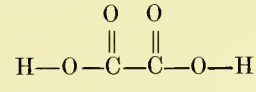
Acids. — If the hydrogen of the aldehyde marked in brackets is replaced by hydroxyl, OH, an acid is produced—



Acetic Acid.

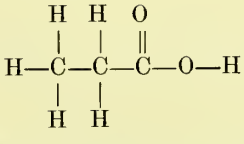
The *carboxyl* group to the right of the dotted line is characteristic of the acids.

The oxidation may be carried on at each end of the line and the *divalent acids* are thus produced



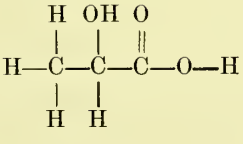
Oxalic Acid.

If in the radical of one of these acids a hydrogen is replaced by hydroxyl, OH, an *oxy-acid* is formed, thus :—

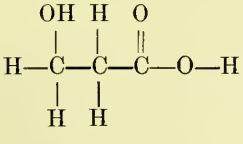


Propionic Acid

may be converted to the two lactic acids called (α) and (β) oxy-propionic acid, according to the carbon which is oxidised



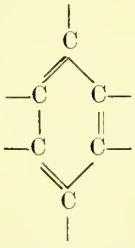
and



Similar oxy-acids are formed from the divalent acids.

Cyclic Compounds

An important series of carbon compounds contains a ring of six carbons, each with an unsatisfied affinity, thus :—



When each hand holds a hydrogen, *Benzene* is formed.

These hydrogens may be replaced by various molecules giving rise to a large series of different compounds.

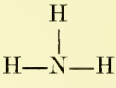
If the ring contain less than six carbon atoms it is called heterocyclic. One of the most important of these is *Pyrrhol*—



which occurs linked to a benzene ring in certain important constituents of the protein molecule.

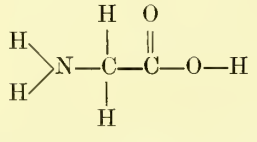
Nitrogen-containing Compounds

Ammonia. — The three-handed nitrogen by linking with three hydrogens forms ammonia,



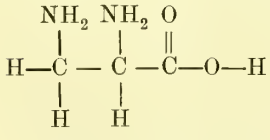
If one of these hydrogens is removed, amidogen, which can link with other molecules, is produced.

Amino Acids.—If one of the hydrogen atoms in the radical of an acid is replaced by amidogen a *mon-amino acid* is formed, thus : —



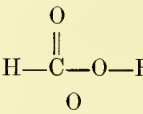
Amino Acetic Acid.

When two hydrogen atoms are thus replaced, a *di-amino acid* is produced—

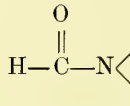


Di-amino Propionic Acid.

Amides.—If the amidogen molecule takes the place of the hydroxyl in the carboxyl of an acid an amide results, thus :—

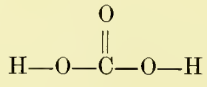


Foramic Acid.

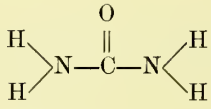


Formamide.

From the divalent carbonic acid—



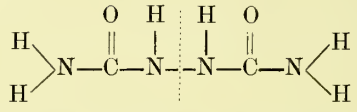
is formed—



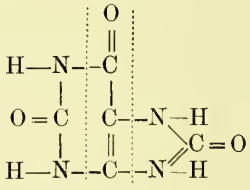
the important substance urea.

Urea molecules may link together—

(*a*) By dropping hydrogens, when biuret is produced—



(*b*) By holding on to an intermediate radical of an acid, *e.g.* an unsaturated three carbon acid. These are diureides, of which the most important is uric acid—



Physiotherapy. — The treatment of disease by physical means, such as exercise, massage, gymnastics, vibration, etc.; mechanotherapy.

Physo-. — In compound words *physo-* (Gr. φῦσα, a bladder or bubble) means bladder-like or related to air or wind; *e.g. physocele* (a tumour or a hernia distended with gas), *physodin* (a neutral substance, with the formula $C_{12}H_{12}O_8$, behaving like a resin, and occurring in the lichen *Parmelia physodes*).

Physometra.—Distension of the uterus with gas, usually as the result of the decomposition of the contents of the uterus (*e.g.* placenta, dead fœtus).

Physostigmatis Semina. *See also* Alkaloids (*Physostigmina*); Eserine; Glaucoma (*Treatment, Myotics*); Pharmacology (*Physostigmine*); Physiology (*Vision, Accommodation*); Toxicology (*Alkaloids*).—The ripe seeds of the

Calabar bean, *Physostigma venenosum*, a tree of West Africa, also known as the Ordeal bean. It contains two alkaloids, *physostigmine* or *eserine*, and *calabarine*, the former being the more important. *Preparations*—(1) Extractum Physostigmatis. *Dose*—$\frac{1}{4}$-1 gr. (2) Physostigminæ Sulphas. Eserine Sulphate. Very soluble in water and alcohol. *Dose*—$\frac{1}{60}$-$\frac{1}{20}$ gr. by the mouth; $\frac{1}{60}$-$\frac{1}{30}$ gr. hypodermically. (3) Lamellæ Physostigminæ. Each contains $\frac{1}{1000}$ gr. of physostigminæ sulphas.

As the extract of physostigmine is somewhat uncertain in composition, and as it contains calabarine, which is in some respects antagonistic to physostigmine, it is advisable always to employ the sulphate of physostigmine when prescribing this drug. Whether given internally or applied locally, physostigmine affects the eye in a manner antagonistic to that of atropine. It causes contraction of the pupil by stimulation of the circular muscular fibres of the iris, diminishes intraocular tension, and causes a temporary increase of the power of accommodation for near objects followed by a spasm of accommodation. It is used in the treatment of corneal ulcerations, and to diminish intraocular tension, as in glaucoma. It has also been employed to prevent the prolapse of the iris after corneal wounds, and to diminish the entrance of light into the eye when photophobia is present. For these purposes the lamellæ, or a solution in water of the strength of $\frac{1}{2}$ to 2 grains to the ounce, may be used. Atropine is more powerful as a mydriatic than eserine as a myotic, and therefore if given to counteract the effect of atropine, physostigmine must be prescribed in somewhat strong solution. On account of its stimulating effect on unstriped muscle, physostigmine is used for atony of the bladder, for chronic constipation, and for bronchitis with emphysema. It is rarely employed for constipation, but in some cases it succeeds when all the drugs have proved inefficient. Unlike cascara, it affects the whole length of the intestinal canal, and, in addition to stimulating the muscular coat, causes an increased flow of secretion. Lastly, because of its inhibitory action on reflex activity and depression of the anterior corneal cells, physostigmine has been recommended in the treatment of tetanus and other convulsive diseases, but only moderate success has followed its administration for this purpose.

Physostigmina and Physostigmine. *See* PHYSOSTIGMATIS SEMINA.

Phytin.—The name given to a substance, said to be an organic phosphorus compound, isolated from the seed of plants (Gr. *φῠτόν*, a plant), and having the formula $C_2H_8P_2O_9$; it is recommended in rickets, osteomalacia, neurasthenia, and wasting diseases, as well as in lactation and convalescence. *See also* PHOSPHORUS.

Phyto-.—In compound words *phyto-* (Gr. *φῠτόν*, a plant) means related to a plant; *e.g. phytalbumin* (vegetable albumin), *phytobiology* (the biology of plants), *phytopathology* (the diseases of plants or the diseases due to vegetable organisms), *phytoteratology* (vegetable teratology), and *phytozoon* (a plant-like animal or zoophyte).

Phytosterin.—A neutral substance obtained from Calabar beans; it is homologous with cholesterin.

Pia or Pia Mater.—The thin, vascular membrane which immediately envelops the brain and spinal cord; the innermost of the three meninges. *See* BRAIN, PHYSIOLOGY OF; MENINGES OF THE CEREBRUM (*Anatomy*, *Tumours*, etc.).

Pia Arachnoid.—The two meninges (pia and arachnoid) considered as one membrane; inflammation affecting both these membranes is called *pia-arachnitis* or *piarachnitis*, or leptomeningitis. *See* INSANITY, PATHOLOGY OF (*Pia Arachnoid*); MENINGES OF THE CEREBRUM (*Inflammations*, *Leptomeningitis*).

Piano-player's Cramp. *See* NEUROSES, OCCUPATION (*Pianoforte Players*).

Piarrhæmia. — The presence of fat (Gr. *πῖαρ*, fat) in the blood.

Piazza's Fluid.—A fluid containing chloride of sodium and chloride of iron; it is used for coagulating blood.

Pica, or Dirt-Eating. *See also* APPETITE (*Perversion*).—Pica (*pica*, a magpie), dirt-eating, or "perversion of the appetite" are names applied to the habit of eating or putting into the mouth various indigestible or disgusting substances. The habit is most frequently noted in hysterical or pregnant women, the insane, imbeciles, and abnormal children.

In some countries dirt-eating is not confined to the above-mentioned classes, as in the Southern States of North America, Africa, and the West Indies, both children and adults are addicted to eating the clay found on the banks of rivers. The same habit is noted in some of the lower animals, as dogs when suffering from worms, and pregnant bitches will eat any filth or garbage.

The capricious appetite of pregnant women is well known, and women who are perfectly normal in their ways at other times will, when pregnant, crunch cinders or dirt between their teeth.

The dirt-eating tendencies of the insane are commonly enough experienced in asylums, and in not a few instances death has resulted from swallowing knives, hair, or stones. Some idiot children will apparently derive pleasure in crawling about the floor and licking the blacking

and dirt off the boots of visitors, or in banging their heads against a wall, or in striking them with their hands.

The most interesting forms of pica may at times be noted in children who cannot be classified as idiots or feeble-minded. In a minor degree an abnormal appetite may be seen in children who are suffering from chronic intestinal catarrh, or who perhaps suffer from worms. The appetite of such will be capricious and uncertain; they will refuse their food at meal-times, but when bathed at night will perhaps take a bite out of a cake of soap with the greatest relish.

In the majority of cases dirt-eating is seen in children with abnormal nervous systems, their eccentricities cropping up in other ways besides in the way of relish for dirt, wall plaster, or the wool off blankets. There may be a tendency to masturbation, moral delinquencies, epileptiform convulsions, or marked perverseness or contrariness of character. The perverted appetite which delights in pushing pieces of slate-pencil up the nostrils, chewing blankets, and crunching cinders, is evidence of a neurosis which ought to put parents and teachers on their guard. While care and careful training will in a proportion of cases result in a more normal state of things, the dirt-eating neurosis marks a tendency to an unbalanced nervous system, which in later years may result in a complete breakdown.

Picein.—A glucoside ($H_{18}O_7H_2O$) obtained from the spruce fir (*Picea excelsa*).

Pick's Bundle.—An abnormal bundle of nerve fibres in the central grey matter of the medulla oblongata; Henle-Pick's bundle. *See* A. Pick, *Arch. f. Psychiat.*, xxi. 636, 1889-90; *Neuraxe*, v. 151, 1903-4.

Picoline.—An organic base or alkaloid (C_6H_7N) obtained from coal-tar; methyl-pyridine.

Picramic Acid.—Picric acid (*q.v.*) in which one NO_2 radicle has been replaced by NH_2, giving the formula $C_6H_5N_3O_5$.

Picric Acid. *See also* Burns and Scalds (*Treatment, Local*); Urine, Pathological Changes in (*Albuminuria Tests*). — Trinitrophenol. Made by dropping phenol into fuming nitric acid. It occurs as yellow crystals. It is soluble 1 in 95 of water and 1 in 10 of alcohol. Picric acid is used extensively in the treatment of burns. It relieves pain, prevents sepsis, and promotes cicatrisation. A watery solution is employed, made up as follows:— picric acid ʒiss; absolute alcohol ℥iii; distilled water ℥xl. Pieces of gauze or lint are soaked in this and applied to the burned surface, and kept in position with wool and a bandage without the addition of any waterproof covering. The dressing should be removed in a few days by moistening the now dry gauze with the same solution, and then a similar dressing is applied again. If too large an area is treated with picric acid there is danger of general poisoning from absorption. Solutions and ointments of similar strength have been recommended for erysipelas, eczema, pruritus, and gonorrhœa. Esbach's reagent, used for the quantitative estimation of albumen in urine, contains picric acid and citric acid in water. A similar solution is said to give excellent results as an application to unbroken chilblains. *Ammonium Picrate*, in doses of ½ to 1½ grains, has been used in the treatment of malaria and of exophthalmic goitre.

Picrocarmine.—A staining agent used in histological investigations, containing carmine, ammonia, and picric acid. *See* Post-mortem Methods (*Preparation of Tissues for the Microscope*).

Picrol.—An antiseptic preparation containing iodine; chemically it is potassium dicodoresorcinmonosulphate.

Picrorhiza.—The rhizome of *Picrorhiza Kurroa*, used as a bitter tonic (dose, 10 to 20 grains) and antiperiodic (dose, 40 to 50 grains); it is officinal in the Indian and Colonial Addendum to the British Pharmacopœia; and there are two preparations, the *Extractum Picrorhizæ Liquidum* (dose, 20 to 60 m.), and the *Tinctura Picrorhizæ* (dose, ½ to 1 fl. dr.).

Picrotoxinum. — Picrotoxine is a neutral principle or glucoside obtained from *Cocculus indicus* (the fruit of *Anamirta paniculata* or Indian fish-berry). *See* Cocculus Indicus. It is used externally (as an ointment) in cases of pediculosis for its antiparasitic action; internally it has been given, in doses of $\frac{1}{100}$ to $\frac{1}{25}$ grain in pill, in epilepsy and in the night sweats of phthisis. It must be given with care, as it is a powerful poison. *See* Toxicology (*Alkaloids, Cocculus Indicus*). Chemically, picrotoxine has been separated into *picrotoxine* proper, $C_{15}H_{16}O_6.H_2O$ (bitter and poisonous), into *picrotin*, $C_{25}H_{30}O_{12} + nH_2O$ (bitter and non-poisonous), and into *anamirtin*, $C_{19}H_{24}O_{12}$ (not bitter, and non-poisonous); but other views as to its chemical composition have been advanced.

Piedra. *See* Skin Diseases of the Tropics (*Piedra*).

Pietrapola. *See* Balneology (*France, Sulphur Waters, Corsica*).

Pigeon Breast. *See* Rickets (*Clinical Features, Chest*). *See also* Deformities (*Rickets*); Nose, Post-nasal Adenoid Growths (*Symptoms, Acquired Pigeon Breast*).

Pigeon Toe. *See* Deformities (*Hallux Varus or Pigeon Toe*).

Pigmentation. *See* Adrenal Glands, Addison's Disease (*Morbid Anatomy*); Alcoholism (*Vaso-motor Variations, Pigmentation*); Drug Eruptions (*Pigmentation*); Liver, Diseases of (*Pigmentation of the Liver*); Nerves, Multiple Peripheral Neuritis (*Epidemic due to Arsenic, Pigmentation*); Pregnancy, Affections and Complications (*Cutaneous Rashes*); Pruritus (*Consequences, Pigmentation*); Rheumatism, Rheumatoid Arthritis (*Clinical Characters*); Sclerodermia (*Circumscribed Form, or Morphœa*); Skin, Pigmentary Affections of the.

Pigmentodermia. *See* Skin, Pigmentary Affections of the.

Pigments of the Body and Excreta.

Introductory 97
The Lipochromes 97
Hæmoglobin and its Derivatives . . 99
The Melanins 100
Some Pigments not included in these Groups 101

See also Blood (*Special Examination, Pigment Granules*); Hæmatoporphyrinuria; Hæmoglobinuria; Physiology, Tissues (*Pigment Cells*); Physiology, Blood (*Red Cells*); Physiology, Food and Digestion (*Bile Pigments*); Physiology, Excretion (*Urine Pigments*); Skin, Anatomy and Physiology (*Pigment of the Skin*); Skin, Pigmentary Affections of; Spectroscope in Medicine (*Classification of Spectra*); Urine, Pathological Changes in (*Pigments*).

Of the pigments which contribute to the coloration of the various organs, tissues, and excreta of the animal body, some perform very important functions, whereas others can only be regarded as mere waste products which are thrown out in the urine and fæces. These latter are for the most part products of the metabolism of the most important of the coloured constituents of the body, namely, hæmoglobin.

The spectroscope has afforded very valuable aid in the recognition and study of many of the animal pigments, and especially of those derived from hæmoglobin, and renders possible their detection even when present in quantities so minute that they could hardly be found by other means. However, its indications require to be controlled by chemical methods, especially when it is sought to obtain the individual pigments in a condition of purity.

Many of the animal pigments owe their importance to the parts which they take in adornment; but it must be remembered that many of the most brilliant hues of birds and insects are not of pigmentary origin, but are optical effects, interference colours, due to the structure of the surfaces which display them. Even excretory products are sometimes utilised for purposes of adornment, as, for example, uric acid, which, as F. G. Hopkins has shown, serves as the white pigment of the wings of certain butterflies, the Pieridæ.

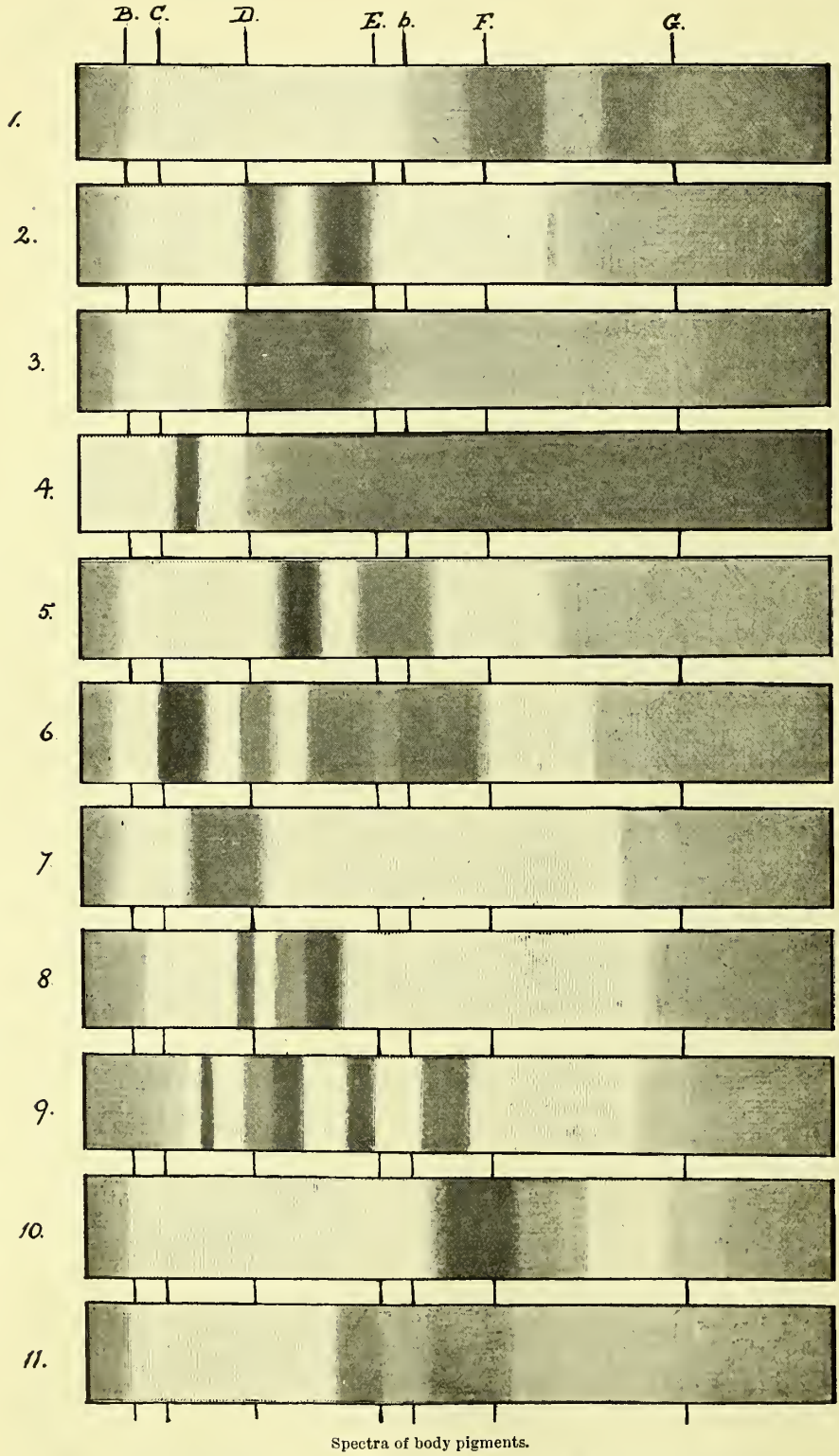
Important as surface pigmentation is in connection with sexual selection, it is no less important in affording protection by approximating the colours of animals to those of the surroundings amongst which they live, or by offering a conspicuous warning that a brilliantly tinted insect belongs to a species which is unsuitable for food. In man such functions of the surface pigments are less conspicuously important than amongst the lower animals, but the colouring matters of the hair and skin, not to mention hæmoglobin itself, which exerts a pronounced influence upon the surface tints, have no inconsiderable share in adornment.

The dark pigment of the choroid performs an important function in rendering the eyeball a dark chamber, and hæmoglobin and the other respiratory pigments met with in some of the invertebrata are second to no other constituents of the animal body in the importance of the work which they perform, quite apart from any ornamental function.

Our knowledge of the animal colouring matters is as yet very imperfect—far too incomplete, indeed, to permit of the satisfactory classification of the entire group. Many of them, however, may be placed in one of three fairly defined groups. One of these embraces the various pigments known as lipochromes. In a second are included hæmoglobin and its various coloured derivatives; and in the third are classed the dark brown or black pigments which have received the name of melanins, and which are by many regarded as hæmoglobin derivatives.

The Lipochromes (No. 1, p. 98), or fat pigments, also known as luteins, are very widely distributed, both in the animal and vegetable kingdoms. They are for the most part yellow or red colouring matters, all of which are soluble in alcohol, ether, and chloroform, and are usually met with in combination with fats, from which they may be separated, without undergoing decomposition, by the process of saponification. The pigments of this class also agree in being more or less rapidly decolorised by light, with the formation of colourless derivatives which, according to Krukenberg, are allied to cholesterin. In the solid state they yield a blue colour with concentrated sulphuric or nitric acid, and some of them yield a blue colour with iodine, whereas with others this reaction is only very imperfectly obtained. The lipochromes are characterised spectroscopically by showing absorption bands towards the violet end of the spectrum, either a single band near the F line, or, in addition, a second band between F and G.

Carotin, the colouring matter of the carrot, has been more thoroughly studied than any other



Spectra of body pigments.

1. Lipochrome.
2. Oxyhæmoglobin.
3. Hæmoglobin.
4. Methæmoglobin.
5. Carboxyhæmoglobin.
6. Acid hæmatin.
7. Alkaline hæmatin.
8. Hæmatoporphyrin (acid).
9. " (alkaline).
10. Urobilin.
11. Uroerythrin.

lipochrome, and this and several other members of the group have been obtained in crystalline form. Husemann assigned to this pigment the formula $C_{18}H_{24}O$, but Arnaud has more recently maintained that carotin, as it exists in the vegetables which it colours, contains no oxygen, and assigns to it the formula $C_{26}H_{38}$. The absence of nitrogen, at any rate, appears to be a feature common to all the members of the lipochrome group.

A lipochrome which has received considerable attention, and which has a wide distribution in the animal kingdom, is tetronerythrin, which was first isolated by Würm from the red areas around the eyes of certain birds, and has been found by Halliburton to be the red pigment met with in the skin, blood, and hypoderm of the lobster and allied crustacea.

Lutein, the pigment of the corpora lutea, was first described by Thudichum, who thus laid the foundation of the study of this group of substances.

When human fat is extracted with ether the yellow solution

which is obtained shows two well-marked absorption bands towards the violet end of the spectrum, due to the lipochrome which it contains, and a similar spectrum is seen when yolk of egg is treated in the same manner. Other interesting members of the group are the chromophanes of the retinal cones of fishes, birds, and reptiles.

The pigment which is mainly responsible for the yellow colour of the blood-serum and of serous effusions is also believed to be a lipochrome. The colouring matter of human serum has received little special study, but from ox serum Krukenberg was able to extract, by means of amylic alcohol, a yellow pigment which showed two absorption bands in the position of lipochrome bands, and Halliburton has obtained similar results with the blood of birds, turtles, and mammalia.

Other pigments may, of course, contribute to the coloration of the serum, such as hæmoglobin, bilirubin, which Hammarsten has shown to be frequently present in the serum of horses, as well as urobilin and other excretory pigments on their way to be excreted by the kidneys.

Hæmoglobin and its Derivatives. — In the present article hæmoglobin claims consideration only as a pigment of the body, and as the parent substance of a large family of coloured derivatives. For descriptions of the chemical and spectroscopic properties of these substances, as well as of the far more important respiratory function of hæmoglobin, the reader is referred to works on physiology.

It is a familiar fact that the respective tints of arterial and venous blood are dependent upon the change of oxyhæmoglobin to reduced hæmoglobin, and *vice versa*. Changes of the colour of the blood are also brought about by the combination of hæmoglobin with gases other than oxygen, as, for example, the cherry tint observed in cases of poisoning by carbon monoxide.

In the surface coloration of the human body hæmoglobin has an important share, and its influence is obvious not only in the visible mucous membranes, but also in the skin itself. An estimate of the part which it plays is readily formed by the observation of patients suffering from extreme anæmia, as contrasted with those who show rosy tints of health; and also from the blue tint of cyanosis, which sometimes attains to so extreme a degree in sufferers from congenital heart-disease. Moreover, when the skin is examined by reflected light the absorption bands of hæmoglobin are clearly visible with the spectroscope.

Methæmoglobin, a compound which has the same percentage composition as oxyhæmoglobin, but in which the oxygen is more firmly held, is often met with in the urine, to which it gives a dark brown or smoky tint. It is not only in hæmoglobinuria that it is present, but also in the ordinary smoky urine of hæmaturia. Hoppe-Seyler stated, indeed, that blood pigment in the urine is always passed in this form, and it is an undoubted fact that blood pigment which has the form of methæmoglobin in the fresh urine may become converted into oxyhæmoglobin on standing, with a corresponding change of tint from brown to pink.

Hæmoglobin also has an important share in the coloration of the solid tissues, partly owing to its presence in the blood contained in their vessels. MacMunn has described a group of pigments allied to hæmoglobin which he calls histohæmatins, the most important of which is myohæmatin, the colouring matter of muscle. He made an elaborate spectroscopic study of this pigment, which he found to be present in the muscles of insects, as well as in those of other invertebrata which have no hæmoglobin in their blood. He also described a modified myohæmatin obtained by artificial gastric digestion of muscle.

On the other hand, Levy maintained that the myohæmatin of MacMunn was not a distinct pigment, but that the spectra observed were due to admixture of hæmoglobin and hæmochromogen, the formation of the latter being due to post-mortem changes.

The most recent investigator in the field is Mörner, who failed to detect in the fresh muscle of dogs and oxen any pigment yielding the spectra described by MacMunn. He observed, however, slight differences between the spectra of the muscle pigment and the blood-colouring matter of the same animal, the absorption bands of the former being slightly shifted towards the red. Similar differences were observed when the corresponding carbonic oxide and methæmoglobin were prepared. On the other hand, the hæmatin from muscle did not differ from that prepared from the blood, and hence he concludes that the difference lies in the proteid components. He proposes for the muscle pigment the name of myochrome.

Hæmochromogen, the coloured constituent of hæmoglobin, which has a very characteristic spectrum, has been detected by MacMunn in the medulla of the suprarenal capsules, but otherwise is not known to figure as a pigment of the body.

Hæmatin has been met with in urine in sulphuric-acid poisoning, and is possibly present in that excretion more frequently than has hitherto been supposed. It is also found in the alimentary canal, being formed by the action of the digestive juices upon hæmoglobin.

Hæmatoporphyrin, an iron-free derivative of hæmoglobin, was known as a laboratory product before its occurrence as a natural pigment was recognised. It has been found by MacMunn in the integuments of certain invertebrate animals, some of which have no hæmoglobin in their blood, and is one of the colouring matters employed in the decoration of the shells of birds'

eggs. In normal human urine it is present in minute traces, and occasionally, especially as a result of sulphonal poisoning, it appears in the urine in much larger amount (*see* art. "HÆMATOPORPHYRINURIA," vol. iv. p. 21). Traces of this pigment are also met with in fæces and bile, and it is more abundantly present in meconium. In a case of sulphonal poisoning it has also been extracted from the blood.

There is as yet no evidence that hæmatoporphyrin is anything but a waste product of metabolism, and the amount excreted appears to be largely dependent upon the condition of the liver.

Hæmatoporphyrin ($C_{16}H_{18}N_2O_3$) is distinguished by the beautiful pink colour of its solutions, and by the distinctive and somewhat complex spectra shown by its solutions under different conditions. The interest attaching to this pigment is greatly increased by the fact that pigments bearing the closest resemblance to it in their spectroscopic properties have been obtained by Church from turacin, and by Schünck and Marchlewski from chlorophyle; but both turacoporphyrin and phylloporphyrin differ from hæmatoporphyrin in their composition. This raises the question how far a practical identity of absorption spectra affords evidence of the identity of the substances which yield them, and, as we shall presently see, the same question arises with regard to the urobilinoid products artificially prepared from bilirubin and hæmatin. On the other hand, such resemblances may fairly be supposed to point at least to a close chemical relationship between the substances from which such allied products are obtained, and probably to the presence in them of a common radicle.

The bile pigments constitute a second class of iron-free hæmoglobin derivatives, and of these bilirubin and biliverdin are by far the most important. Bilirubin has the formula $C_{16}H_{18}N_2O_3$, and will be seen to be isomeric with hæmatoporphyrin. In the amorphous state it appears as a yellowish red powder, insoluble in water, very slightly soluble in alcohol and ether, but readily dissolved by chloroform. It forms readily soluble compounds with alkalies, and alkaline solutions turn green on standing, owing to the oxidation of bilirubin to biliverdin ($C_{16}H_{18}N_2O_4$). Unlike bilirubin, biliverdin is readily soluble in alcohol, and insoluble in chloroform. Neither of these pigments shows any spectroscopic absorption bands, but merely general absorption of the more refrangible rays.

The evidence of the derivation of the bile pigments from hæmoglobin is of various kinds, such as their increased formation when excessive hæmolysis is going on; the formation of crystals of hæmatoidin, which is apparently identical with bilirubin, in the seats of blood extravasations; and the fact that under the influence of reducing agents both bilirubin and hæmatin yield urobilinoid products.

The bile of the herbivora is green from the presence of biliverdin, but some doubt exists as to the nature of the pigment of normal human bile. Post-mortem human bile is usually golden or brown in tint, and in gall-stones bilirubin calcium is the chief pigmentary ingredient, whereas fistula bile is nearly always of a bright green colour. When the bile pigment traverses the intestine unchanged it may appear in the fæces, either as bilirubin or as biliverdin. When it becomes diffused through the tissues in jaundice it appears in the skin as bilirubin, but in cases of long-standing jaundice a greenish tint develops. In the urine of jaundiced persons either or both of these pigments may be present, and especially in the urine of infants bilirubin may be deposited in crystalline form.

The other known bile pigments, such as bilifuscin, bilihumin, and biliprasin, have been chiefly met with as constituents of gall-stones. Bilicyanin has also been found in gall-stones, and cholhæmatin is a pigment with a characteristic spectrum which has been found by MacMunn in the bile of sheep and oxen.

Urobilin is also found in the bile, and appears to be one of a group of allied pigments which may be prepared by the action of reagents upon the blood and bile pigments. It is characterised by a broad dark absorption band near the F line, and by yielding a green fluorescence with zinc chloride and ammonia.

It is abundantly present, chiefly as a chromogen, in normal fæces, and is probably formed by the action of intestinal bacteria upon bilirubin. It differs, however, in composition from hydrobilirubin, the very similar pigment obtained by the action of sodium amalgam upon bilirubin. It is also met with in urine in traces in health, and here in the form of chromogen, and in far larger amount in many morbid urines, which may show its absorption band with great distinctness. There are good grounds for believing that the urobilin of the urine and the bile are alike derived from the intestinal supply, and it disappears from both urine and bile when complete occlusion of the common duct prevents the entry of bile into the intestine.

Among the derivatives of hæmoglobin a place should probably be assigned to urochrome, the yellow colouring matter of normal urine, which has no distinctive spectrum. From this pigment a urobilinoid substance is obtained by the action of aldehyde upon an alcoholic solution, but this, like the products prepared from hæmatin and bilirubin, exhibits differences from the natural urobilin of the urine and fæces. As to the mode and place of origin of urochrome no further evidence is as yet forthcoming.

THE MELANINS.—Our knowledge of the dark brown and black pigments known as melanins is as yet very imperfect. Numerous analyses

of such pigments have been carried out, but the results which have been obtained differ somewhat widely, even with materials derived from the same source. The differences observed are specially marked as regards the presence or absence of iron and the proportion of combined sulphur. They are, doubtless, in part due to the difficulty of isolating substances of this description unaltered, but yet in a condition of purity, and in part, perhaps, to the presence of more than one pigment of the group in the same situation. Some observers regard the melanins as derivatives of hæmoglobin, basing their opinion in part upon the presence of iron; but others, including Nencki, take a different view of their origin, and Krukenberg looks upon them as more nearly related to the lipochromes than to hæmoglobin.

In the human body melanins play important parts in the pigmentation of the hair and eyes, as well as of the skin of the dark races, and even in the white races in the coloration of moles. In cases of generalised melanotic tumours large quantities of a pigment or pigments of this class are formed, and may be excreted in part in the urine.

The dark pigment of hair has been investigated by Sieber, who found it to be free from iron, but to contain sulphur. Abel and Davis also failed to find iron in this pigment as well as in that of the skin of negroes.

The black pigment of the retina known as fuscin has been found by Kühne and Mays to contain iron, whereas Scherer and Sieber found neither iron nor sulphur in the very similar if not identical colouring matter of the choroid coat of the eye.

The melanins of melanotic tumours have been repeatedly investigated. Heintz found no iron in them; Dressler found no sulphur, but a small quantity of iron was detected in the ash. The pigment isolated by Berdez and Nencki, to which they assigned the name of phymatorhusin, was insoluble in water, alcohol, and ether, but readily soluble in alkalies, from which it could be reprecipitated by acids. No iron was present in it, but it contained sulphur in considerable amounts. From melanotic growths in the horse they isolated a second pigment to which they gave the name of hippomelanin.

Mörner, on the other hand, obtained from human tumours, and from the urine of the patient, two pigments, one soluble and the other insoluble in acetic acid. Both contained iron as well as sulphur. He ascribes the absence of iron from the products isolated by previous observers to the employment of hydrochloric acid in their preparation. The sarcomelanin isolated by Schmiedeberg also contained iron, and similar results have been obtained by Brandl and L. Pfeiffer.

The dark pigment present in the urine in such cases is in part or whole passed as a chromogen, melanogen, and darkening may only take place after exposure to the air. Sometimes, however, the urine is of a very dark colour when passed.

Occasionally, as in a case recorded by Senator, the pigment is present in the blood and ascitic fluid as well as in the urine.

Other Pigments.—By no means all the pigments of the human body can be placed in one or other of the above groups.

For example, the chemical relationships of the visual purple, the unstable pigment of the retinal rods, are unknown. Our knowledge of this substance is largely due to the researches of Kühne.

It is so readily decolorised by light that its separation has to be carried out by the light of the sodium flame. The best solvent is a dilute solution of bile salts, and it can be reprecipitated by removing the bile salts from the liquid by dialysis. It is not only decolorised by light, but also by heating its solutions, by alkalies other than ammonia, by acids, and even by alcohol, ether, and chloroform. The reddish purple solution of the pigment shows no definite absorption bands.

Another pigment which cannot as yet be assigned to any definite class is uroerythrin, the urinary colouring matter which is responsible for the reddy orange tint of febrile urines and that of sufferers from hepatic diseases, and which colours uratic sediments pink. It is an extremely unstable substance, its solutions are rapidly decolorised by light, and it is destroyed by alkalies with the formation of a green product. Its spectrum consists of two feeble bands in the green and blue united by a dark shading.

The indigo pigments are derived indirectly from indol, which is formed in the intestine by the bacterial decomposition of proteids. From the intestine it is absorbed and excreted by the kidneys in the form of indoxyl-sulphuric and indoxyl-glycuronic acids. Indigo blue is sometimes spontaneously deposited from the urine, and in a very few instances renal calculi composed of indigo have been met with. Indoxyl-sulphuric acid is often present in the urine in considerable amount, as, for example, in cases of intestinal obstruction, and under such circumstances the urine, when boiled with hydrochloric acid and a trace of bleaching powder, yields an abundance of indigo blue and red.

Urorosein is a pink pigment which is also obtained by the action of hydrochloric acid upon the urine, from which its colourless chromogen has been isolated in crystalline form. It also is very probably of intestinal origin.

The brown oxidation products of the dioxybenzenes are also occasionally met with in urine, as in cases of carboluria, and in the rare condition known as alkaptonuria.

Lastly, bacterial pigments must be mentioned, such as pyocyanin, which is the product of the

bacillus pyocyaneus, and which occasionally imparts to pus a green colour.

In the present article it has only been possible to give a mere outline of the subject of the pigmentation of the human tissues and excreta, and the limits of space have precluded more than incidental reference to the colouring matters of the lower animals, which present many problems of great interest. Of some of these pigments copper is an essential constituent, as, for example, hæmocyanin, the respiratory pigment of the blood of some crustacea and molluscs. Turacin, the red pigment of the wing-feathers of the touracos, is another copper-containing pigment of much interest. Its properties and composition have been investigated by Professor Church, and, as has been already mentioned, by the action of sulphuric acid upon it a product called turacoporphyrin is obtained, which, in its spectroscopic features, bears the closest resemblance to hæmatoporphyrin.

Pigmentum.—In Pharmacy *pigmentum* is the name given to a preparation which is meant to be painted on the skin or on a mucous membrane.

Pigodidymus.—A teratological type in which two fœtuses are united by the gluteal regions.

Pigs. *See* Anthrax (*In the Lower Animals*); Rheumatism, Chronic (*Comparative Pathology, Pigs*).

Pigstyes.—According to Sanitary Law (L.G.B. Model By-Laws) swine must not be kept within, say, 100 feet of any dwelling, and care must be taken that water for drinking or dairy purposes or for manufacturing drinks be not contaminated by them. According to the Public Health (Scotland) Act of 1897, the Local Authority may make by-laws regarding the construction, situation, and mode of cleansing of pigstyes.

Piles. *See* Rectum, Diseases of (*Hæmorrhoids*). *See also* Hæmorrhoids *and Cross References*; Hydropathy (*Subacute and Chronic Diseases, Piles, Ascending Douche*); Liver, Diseases of (*Functional Disease, Piles*).

Pillars.—The name given, in Anatomy, to supporting structures or columns, such as the pillars of the diaphragm, abdominal ring, fauces, and fornix.

Pills or Pilulæ. *See* Prescribing (*Methods of Administering Drugs, by the Mouth*). *See also* under the various drugs, *e.g.* Aloes, Ferrum, Mercury, etc.

Pilo-.—In compound words *pilo-* (Lat. *pilus*, a hair) means relating to hair; *e.g. pilomotor* (producing movement of hairs), *pilocystic* (of the nature of a dermoid cyst), *pilose* (hairy).

Pilocarpine. *See* Jaborandi; Alkaloids; Glaucoma (*Treatment, Myotics*); Physiology, Excretion (*Sweat Secretion*); Toxicology (*Alkaloids and Vegetable Poisons, Pilocarpine*).

Pilonidal.—Serving as a nest (*nidus*) for the accumulation of hairs, *e.g.* in a cyst or fistula.

Pilosis.—Abnormal development of hair. *See* Hirsuties.

Pimaric Acid.—An acid resin, $C_{20}H_{30}O_2$, contained in the turpentine of *Pinus maritima*.

Pimelo.—In compound words *pimelo-* (Gr. πιμελή, fat) means fatty; *e.g. pimeloma* (a fatty tumour), *pimelorrhœa* (a fatty discharge), and *pimeluria* (fat in the urine).

Pimenta.—Pimento is the unripe fruit of *Pimenta officinalis*, the all-spice tree; it contains a volatile oil (*Oleum Pimentæ*) which contains eugenol, and is given in doses of ½ to 3 m.; there is an *Aqua Pimentæ* (dose, 1 to 2 fl. oz.); pimento has the same pharmacology and therapeutics as cloves.

Pinard's Sign.—Pain on pressure over the fundus uteri during the last three months of pregnancy is said frequently to indicate a breech presentation.

Pineal Body or Gland. *See* Brain, Physiology of.—The pineal gland is a small, fir-cone-shaped body which lies on the dorsal aspect of the mesencephalon between the two upper corpora quadrigemina; it has a hollow peduncle and consists of rounded and tubular follicles; it is a rudimentary structure in man, but in some vertebrates it is more highly developed and resembles in them an invertebrate eye (pineal eye). A pineal tumour causes symptoms. *See* Auditory Nerve (*Diagnosis of Nerve Deafness*).

Pinene.—A hydrocarbon ($C_{10}H_{16}$) or terpene found in the oil of turpentine and other oils.

Pine Oil.—Pine oil or *Oleum Pini* is obtained from *Pinus pumilio*, and is known also as *pinol*; it produces the same effects as oil of turpentine (*vide* Turpentine), and is used as an inhalation in laryngitis, bronchitis, etc.; it has been recommended also in rheumatism and tuberculosis.

Pineoline.—A proprietary preparation, used externally in skin diseases, and said to contain pine oil combined with vaseline and lanoline.

Pinguecula or Pinguicula.—A small tumour or yellow patch situated on the ocular conjunctiva, usually on each side of the cornea. *See* Conjunctiva, Diseases of (*Various Conditions, Pinguecula*).

Pinic Acid.—A constituent of brown resin, having the formula $C_{20}H_{30}O_2$, and yielding, if cautiously heated, colophonic acid.

Pink-eye.—A sort of influenza in horses. *See* Influenza (*Historical*).

Pinna.—The external ear or auricle. *See* Ear, External, Diseases of (*Anatomy*).

Pinocytosis.—The power believed to be possessed by phagocytes of imbibing liquids and so rendering them harmless; the word is derived from the Greek πίνω, I drink, and κύτος, a cell or jar.

Pinol. *See* Pine Oil.

Pinta.

Definition.—A tropical disease of the skin, characterised by the development of variously coloured patches which later become scaly.

The chief countries in which the disease is frequently met with are:—Brazil, Mexico, (especially the provinces of Michoacan and Guerrero), Central America, Venezuela, Colombia, and Peru. It is particularly common in the neighbourhood of creeks and marshes, and is never found at a greater altitude than 2000 metres.

It affects all races, but more especially the half-breeds and those in poor circumstances, while the pure negro seems to enjoy a partial immunity.

Symptoms.—The disease begins as one or more small patches, usually situated on the face or extremities, seldom on covered parts. The colour is at first usually slightly red from hyperæmia in white subjects, but in blacks the patch may be fawn-coloured from the first.

The patches rapidly become scaly, and then develop gradually their characteristic colour. This may be a blue or a black in the more superficial varieties, or a red or white in the deeper form. New patches continue to form, while the old ones spread centrifugally, until eventually the whole body with the exception of the palms and soles may be affected. Pruritus is almost invariably severe, and may be noticed even before the appearance of the skin changes.

As the patches become older the skin undergoes more or less degenerative changes, hair turns white and falls out, and the skin shows hard papules, rugosity, and general sclerosis. Probably most of these later changes are due to scratching, since most writers agree that in the chronic widespread cases the itching becomes intense. In the deep variety there is occasionally a discharge superadded, and in the flexures of joints troublesome ulceration may occur. In cases where discharge or ulceration occurs the patient acquires a disagreeable odour, which has been likened to the smell of dirty linen or cat's urine. There are no constitutional symptoms other than those induced by the continual itching, and the disease may, if untreated, persist during the whole lifetime of the patient without serious consequences.

Etiology and Pathology.—There still exists some difference of opinion as to the nature of the disease, Lier, of Mexico, maintaining that it is not contagious, after numerous failures in the attempt to inoculate it, and therefore considering that it is rather to be regarded as an abnormality of pigmentation due to attempted adaptation of the skin to the tropical climate. Gastambide, on the other hand, described several varieties of fungus belonging to the order of moulds (penicillium), which he found present in the horny layer in the blue and black varieties, and in the deeper layers of the epidermis and corium in the red and white varieties. The fungi varied in the different colours, and Gastambide points out that though the same patient may exhibit several colours at once in different patches, yet a patch of one colour never develops into one of another. The majority of observers seem to agree more or less with Gastambide's views, regarding it as contagious and due to a mould. Inoculation may occur directly from another patient, or possibly by the bites and stings of insects.

Some confusion still seems to exist as to the relation of the disease to those described under the names of Caratés and Cute. Most writers assume that they are identical, but Barbe insists that they are all three separate affections, and says that they may be distinguished by the following characteristics:—Caratés is a slowly progressive disease, taking three years to reach its period stage of full development; pinta is much more rapid, may develop in forty-eight hours, and is extremely contagious; cute is also a disease of rapid development, and differs from both the others in that it is accompanied by fever.

No difficulty should be found in making the diagnosis in patients who come from the affected area. From leprosy the distinction could easily be made by the absence of the characteristic anæsthesia, from Addison's disease by the absence of marked asthenia and abdominal crises, from tinea versicolor, erythrasma, and trichophytosis by the examination of the scales.

The treatment is that of other epiphytic diseases of the skin, *e.g.* unguentum chrysarobini, unguentum hydrargyri nitratis, linimentum iodi. Long-established cases require great persistence in the treatment, as the disease is extremely liable to recur.

Pinus. *See* Pine Oil.

Pin Worms. *See* Parasites (*Nematodes, Oxyuris Vermicularis*).

Pioscope.—A lactometer invented by Heeren for testing the purity of milk by its colour (Gr. *πίων*, rich or oily).

Piperazine.—The chemical formula of this substance is $C_4H_{10}N_2$, and it is formed by the action of sodium glycol on ethylenediamine hydrochloride. It occurs in small colourless crystals which are very hydroscopic. It has been introduced for the treatment of the "uric-acid diathesis," because, theoretically, it should form with uric acid a urate of piperazine which is soluble and easily eliminated. Clinical reports as to its efficiency have been very contradictory, some stating that it increases the output of urea and diminishes that of uric acid, others that no beneficial effect is produced. It is prescribed in 5-grain doses three times daily, and should be given in solution, or as "effervescent piperazine," which contains 5 grains to each drachm. It has been recommended to prevent the formation of uric acid calculi, and to diminish irritability of the bladder when this is due to excess of uric acid in the urine. *Lycetol*, *Lysidine*, and *Sidonal* are substances for which similar actions are claimed.

Piperidine. *See* Pepper.

Piperine. *See* Pepper.

Piper Nigrum. *See* Dermatitis Traumatica et Venenata (*Casual Agents, Vegetable, Piper Nigrum*); Pepper.

Pipette.—A glass tube open at both ends, but generally narrower at one end than at the other, used for the transference of definite quantities of fluid from one place to another.

Pirogoff's Operation. *See* Amputations (*At Ankle-joint, Pirogoff's*).

Piroplasma.—An organism belonging to the Hæmosporidia group of the Sporozoa, giving rise to the diseases known as *piroplasmoses* (*e.g.* "spotted fever" in man and "red water" in cattle). *See* Parasites (*Protozoa, Piroplasma*); Dum-Dum Fever; Kala-azar.

Pisciarelli. *See* Balneology (*Italy, Chalybeate Waters*).

Pisiform.—Shaped like a pea (*Lat. pisum*, a pea), *e.g.* the pisiform bone of the carpus.

Pitch. *See* Pix. *See also* Physiology, Respiration (*Voice, Pitch*).

Pithecanthropus Erectus.—The "missing link," or pithecoid (ape-like) ancestor of man. *See* Anthropology.

Pitlochry. *See* Therapeutics, Health Resorts (*Scotch*).

Pitot's Tubes.—In sanitary engineering these tubes are used to determine the velocity of rivers: they are tubes open at both ends, but having one end about at right angles to the rest of the tube; the water stands in the tubes at different levels according to the way in which the bent end faces or is turned away from the current, and by comparing the levels in differently placed tubes the velocity of the stream can be deduced.

Pitting. *See* Smallpox (*Treatment, Prevention of Pitting*).

Pituita.—Phlegm, or the secretion from a mucous membrane.

Pituitary Body (Hypophysis cerebri). *See also* Acromegaly (*Pathogenesis*); Brain, Physiology of; Brain, Tumours of (*Morbid Anatomy*); Physiology, Internal Secretions or Hormones (*Pituitary Body*).

Structure.—This body consists of two lobes of different developmental origin: (1) An anterior larger lobe, glandular in type, derived from the embryonic oral cavity; (2) a posterior smaller lobe, belonging genetically to the brain, a continuation of the infundibulum.

The anterior epithelial lobe (hypophysis proper) is light reddish-brown, darker in colour than the posterior one, and in its origin from the oral epiblast is homologous with the buccal glands. It is of great interest histologically, as it has a structure in some respects resembling that of the thyroid. It is made up of numerous tubes, which may be only slightly convoluted, and which are arranged in a network. These tubes are held together by connective tissue strands in which there run numerous blood-vessels of wide lumen and with delicate walls. There are also lymphatics between the tubules, which gradually unite to form a network in the capsule. The cells which line the tubes are of two kinds, the one set being larger and more granular than the other. They may contain degenerated red cells or blood pigment, and, according to Benda, fat in the form of globules. A colloid substance is frequently met with in the tubes in the blood-vessels and also in the connective tissue spaces (lymphatics). Fine nerve fibres, sympathetic in origin, have also been described by Berkeley. There have been numerous cases described where the colloid material has occurred in cysts in this lobe, and enlargement of the pituitary has often been noted in cases of acromegaly.

The posterior lobe arises as a hollow downgrowth from the forerunner of the third ventricle; but in the higher vertebrates it is very small and atrophied, consisting mainly

of bundles of connective tissue with branched pigmented cells and smaller clasping cells free from pigment. Its original nervous structure is entirely lost.

Functions.—Recent investigations have shown that the pituitary, like the suprarenal and thyroid glands, probably furnishes an internal secretion which is of importance in maintaining the normal metabolism. The three methods of investigation which have been employed in attempting to arrive at a knowledge of the character of internal secretions of other ductless glands—namely, that of the *clinician*, where the effects of disease are noted, that of experimental removal, and lastly that of injection of extracts —have all been made use of. The results arrived at, although very valuable, are still not sufficiently definite to warrant one in going further than stating the probable rôle of the body. It is exceedingly unlikely that the pituitary acts in a similar way to the thyroid, although Rogowitsch has stated that in rabbits it enlarges after removal of the thyroid, thus supplying a vicarious secretion, and others have described hypertrophy, tumour growths, etc., in cases of myxœdema. The relationship between enlargement of the pituitary and the occurrence of acromegaly is a more probable one, but their simultaneous occurrence is by no means constant. The experiments of Schiff on excretion of nitrogen and phosphorus, after feeding with pituitary extracts, do not warrant one in drawing the conclusion that the extracts exert an influence on bone metabolism. After complete removal of the gland, animals only survive for a comparatively short period, dying usually within a fortnight. The body temperature is lowered, the animals gradually become feebler, suffer from muscular tremors, and afterwards spasms of a severe nature, and finally pass into a dyspnœic condition. More exact information was to be expected from investigating the effects of injection of pituitary extracts, and these expectations have been fulfilled by the work of Schäfer and his collaborateurs. The action is quite different from that possessed by the thyroid, as aqueous extracts of the infundibular or nervous portion have a very marked effect upon the blood pressure, the resultant rise being due not only to an increased heart's action, but especially to a direct local action upon the systemic arterioles. This contraction of the arterioles can be demonstrated by circulating a normal saline extract of the pituitary through the blood-vessels of a frog after destruction of its entire nervous system, or by the plethysmographic method applied to the limbs, intestines, or spleen, when in every case there is a distinct diminution in volume. Unlike the suprarenal, however, the aqueous infundibular extract produces, after a brief latent period, a marked expansion of the kidney, accompanied at the outset only by a slight rise in general blood pressure which lasts only a few minutes. This expansion continues for a long period, and concomitant with it there is a marked diuresis, which dies away, however, before the expansion ceases. The substance or substances which produce this effect are not soluble in alcohol; in fact saline extracts of the alcohol soluble constituents of the glands seem to produce in some cases at least the opposite effects—temporary fall in blood pressure, contraction of the kidneys, and diminution in flow of urine. This action is probably, however, not specific, but due rather to depression of cardiac activity, a condition easily brought about in the cat, in which animal the above-mentioned effects are most easily produced. So far, then, as our present knowledge leads us, the specific action of the infundibular part of the pituitary seems to be, in part at least, a renal one. The nature of the active principle or principles requires still to be worked out.

Pityriasis.—A scaly or scurfy condition of the skin (Gr. πιτυρίασις, scurf) characteristic of several cutaneous affections. *See* Eczema (*Mycosiform, Seborrhœic Variety*); Lung, Tuberculosis of (*Complications, Integumentary, Pityriasis tabescentium*); Nails, Affections of (*In Pityriasis rubra*); Pregnancy, Physiology (*Changes in the Skin*); Psoriasis (*Diagnosis*); Skin, Parasites (*Tinea Versicolor*); Skin, Pigmentary Affections of (*Spurious Pigmentation in Pityriasis Versicolor*).

Pix Burgundica. — Burgundy pitch, derived from the Norway spruce, a plant of Europe and Asia. It is a hard, resinous exudation of a dull red colour, and its chief constituents are pimaric acid and a volatile oil. It melts at the temperature of the body, and is useful for making plasters. In itself it is mildly stimulant to the skin, and is used as a counter-irritant for muscular rheumatism, bronchitis, and sprains. *Preparation—Emplastrum Picis.*

Pix Carbonis. *See* Tar.

Pix Liquida. *See* Tar.

Pixol.—A solution of wood-tar in soap, with caustic potash.

Pjätigorsk. *See* Balneology (*Russia, Caucasus*).

Placebo.—A medicine "adapted more to please than benefit the patient"; literally the word means "I shall be pleasing"; a familiar form of the placebo is a bread pill.

Placenta. *See* Abortion (*Varieties of Product, Placental Causes*); Ectopic Gestation (*Treatment, Removal of Placenta*); Fœtus and Ovum, Development of (*Placenta, Development of, and at Term*); Labour, Stages and Duration (*Third Stage*); Labour, Diagnosis and Mechan-

ism (*Transverse Lies and Placenta Prævia*); Labour, Management of (*Third Stage*); Labour in Multiple Pregnancy (*Management of Third Stage*); Labour, Accidental Complications (*Retention of Placenta*); Labour, Injuries to the Generative Organs (*Acute Inversion of the Uterus, Causes*); Molar Pregnancy (*Placental Polypus*); Pregnancy, Multiple (*Placentæ and Membranes*); Pregnancy, Pathology of Diseases of Placenta and Cord (*Inflammation, Hæmorrhage, Œdema*, etc.); Pregnancy, Pathology of, Intra-Uterine Diseases (*Transmission of Microbes through Placenta*, etc.); Pregnancy, Pathology of Hæmorrhage (*Accidental, Unavoidable, Rupture of the Uterus*); Puerperium, Pathology (*Pyæmia, Causes*); Puerperium, Pathology (*Deciduoma Malignum*); Teratology (*Placental Parasites*).

Placentation.—The mode of formation of the placenta.

Placentitis.—Inflammation of the placenta. *See* Pregnancy, Pathology (*Diseases of the Placenta*).

Placentoma.—A tumour derived from a portion of the placenta or of the decidual membrane which forms the placenta; malignant placentoma is a synonym of chorionepithelioma or deciduoma malignum.

Placido's Disc. *See* Cornea, Affections of (*Conical Cornea, Diagnosis*); Eye, Clinical Examination of (*Examination of Cornea, Kerato-conus, Diagnosis*).

Pladarosis.—A soft tumour or wart of the conjunctival surface of the eyelid (Gr. πλαδαρός, moist).

Plagiocephaly. — An obliquely deformed head, due to asymmetrical development (Gr. πλάγιος, slanting).

Plague.

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See also Balneology (*Historical, Baths for Plague*); Epidemiology (*Seasonal Relations of Plague*); Gangrene (*Varieties, Infective*); Glandular Fever (*Diagnosis*); Immunity (*Specific, Plague*); Leucocytosis (*In Infective Conditions*); Milk (*Pathological*); Pneumonia, Bacteriology of (*Plague associated with Pneumococcus*); Pneumonia, Clinical (*Etiology, Plague*); Post-Mortem Methods (*Bacteriological Investigations, Plague*); Purpura (*Symptomatic, Infective*).

1. Definition. — Plague is an acute infectious disease, caused by a specific bacillus and characterised by inflammation of the lymphatic glands, sometimes by pneumonia or septicæmia.

2. Synonyms.—Pestis, Pestilentia, Pestis bubonica, Clades inguinaria glandularia, Oriental Plague, Indian Plague, Pali Plague, Black Death, Die Pest, Beulen Pest, Peste, Pestilenza, Tschuma, Mahamari, and a great many other names, some of which signify "epidemic disease with great mortality," "the rat disease," etc. etc.

3. History and Geographical Distribution. —The history of the distribution of plague epidemics divides itself into two portions: epidemics before and after 1894 A.D., in which year, in the outbreaks in Southern China, the present widely diffused series of plague epidemics commenced.

Regarding the early portion of plague history little need be stated. In the writings of Rufus of Ephesus, reference is made to an epidemic of a disease in Egypt, Libya, and Syria characterised by fever and buboes, about the beginning of the second century B.C. About the beginning of the Christian era another plague epidemic was noted in Libya by two Alexandrian physicians. We find no other certain reference to plague until the occurrence of the great *Justinian Plague* in the sixth century A.D., which ravaged the whole Roman Empire "to the ends of the habitable world." This lasted fifty or sixty years, and wrought most frightful havoc wherever it appeared. From the second half of the sixth till the fourteenth century many severe epidemics of plague harassed Europe. The *Black Death* of the fourteenth century is the next epidemic of plague of which we have much

information. It is said to have started in Eastern Asia, and to have spread to Asia Minor, Northern Africa, and Europe, reaching the Crimea in 1346; by 1349 and 1350 it had overrun the whole continent of Europe. Hecker estimates the loss of life in this pandemic at 25 millions, about a quarter of the then population of Europe. Plague, with which is probably included several other epidemic diseases, continued to be prevalent in Europe throughout the fifteenth, sixteenth, seventeenth (in which occurred the *Great Plague of London*), and eighteenth centuries, sometimes affecting small areas, at other times in the form of a pandemic.

Towards the latter part of the seventeenth century plague appeared to be gradually retreating from Europe, and from the middle of the eighteenth century only the south-eastern parts of the continent were permanent seats of the disease. In the nineteenth century plague was epidemic in this portion of the continent from time to time till 1841, when, with the exception of an outbreak in 1879 in Astrakhan on the borders of Asia, plague disappeared from Europe till the localised epidemics which have occurred during the last few years.

Several *endemic centres of plague* have been recognised throughout the world, most of them for a long period, the chief of them being as follows:—

(1) The district of *Assyr*, in Arabia, on the eastern shore of the Red Sea.

(2) Portions of *Persia and Mesopotamia.*

(3) Southern China, especially the province of *Yun-nan* on the Tibet frontier.

(4) The districts of *Kumaon and Garhwal*, Upper India, on the southern slopes of the Himalayas.

(5) The district of *Kisiba* (German East Africa), and an adjoining locality situated in the south of the province of Uganda, to the west of Lake Victoria Nyanza.

(6) The *Transbaikal Province*, Eastern Siberia, and Mongolia.

(7) More recently Dr. Thiroux has added the *Island of Réunion*, where he alleges that plague, locally known as *Lymphangite infectieuse*, has existed in a mild form for many years.

The present history of plague distribution starts from the year 1894, when the disease assumed a new phase. Previous to this, plague had for a long period been confined to the nomadic tribes in Northern Persia and Central Asia, the Arabs in Assyr, and the Bedouins in Mesopotamia. The fresh characteristic which plague then assumed was to attack large and populous cities in Southern China and India, such as Canton, Hong-Kong (1894), and Bombay (1896). Travelling down to Canton from Yunnan, near Tibet, the disease spread to Hong-Kong and thence to Bombay. From Hong-Kong and Canton many parts of China have become infected, and from Bombay plague has spread over a considerable portion of the Indian peninsula; in 1902 the epidemic wave extended over districts in India never before infected during the present pandemic, and as many as 80,000 cases and 61,000 deaths from plague have been reported from the Punjab in a single month (March). Since 1897 Japan, the Philippines, Honolulu, Madagascar, British East Africa, Aden, Mauritius, Australia, South America, and South Africa have suffered, and nearer home Oporto, Alexandria, Port Said, as well as other towns, have become infected. Cases are continually being reported from the Levantine cities, and these, together with the repeated cases of the disease occurring on board ships arriving in European ports, make plague a very distinct danger to this country. Localised outbreaks, fortunately on a very small scale, have already occurred in Glasgow and Liverpool, but were soon suppressed by the authorities. The geographical distribution of plague at the present time, together with a history of recent epidemics, is considered at length in a special plague volume just issued by the Medical Officer to the English Local Government Board.

4. Modes of Entry and Means of Spread

A. Modes of Entry.—Plague may enter the body in three ways, by—(1) Inoculation; (2) Inhalation; (3) Ingestion.

(1) *Inoculation.* — The commonest mode of entry of the plague bacillus into the body is by the skin. This probably does not take place through unbroken skin, but through abrasions of various sorts, such as wounds, scratches, etc., and the bites or stings of insects. It is not necessary to suppose that the insect itself introduces the infection, but it is probable that this may enter through the bite, either by being already on the skin when the insect bites, or by subsequent contamination of the wound. The irritation of the bite may sometimes produce scratching, which may be done with an infected finger, and in this way inoculation may occur. The rings worn on the toes in India and elsewhere, which are often associated with the presence of sores on the toes, may create a channel of entry for plague infection. In connection with this fact, however, it must be noted that it is the inguinal and femoral glands of those, such as Europeans, who usually go about booted which are chiefly affected.

The Indian Plague Commission, in investigating this point, found that there was a relation between the area of skin surface draining into a particular set of glands and the frequency with which those glands were affected. They found that while the areas of skin surfaces which drain respectively into the neck, axilla, and groin stand to one another approximately as 1 : 1·8 : 5, the hospital statistics at their disposal showed that the relation between the numbers of primary buboes in the cervical,

axillary, and inguinal regions was 1 : 1·3 : 5·8. The explanation therefore of the more frequent occurrence of buboes in the groin seems to be that a much larger skin surface drains into these glands, and therefore the greater the skin surface the greater the chance of infection of a region. As instances of infection by inoculation may be cited those cases infected at post-mortems by cutting the fingers, or through abrasions already present. In such cases a bubo subsequently develops corresponding to the site of inoculation. The frequency of buboes in the neck in children may be ascribed to their habit of putting things into the mouth, the channel of entry being cracked lips, or other wounds in that neighbourhood; or perhaps infection reaches the neck through the tonsil. There is a case recorded of a pneumonic patient spitting on to the conjunctiva of a nurse, who developed a cervical plague bubo. The site of inoculation is sometimes found as a small slightly inflamed papule or pustule, and the plague bacillus may in some cases be recovered from the point of entry. The glands corresponding to this pustule are those affected, and sometimes inflamed lymphatics may lead from the pustule to the bubo. As a rule one is unable to discover definitely the point of inoculation.

(2) *Inhalation.*—This is probably the mode of entry of infection in pneumonic and sometimes septicæmic plague. It is well recognised that exposure to an ill-ventilated atmosphere, charged with plague bacilli floating in the dust of such a room, is a common source of infection. This latter condition appears to have been the mode of infection at the "Wakes" held in the early Glasgow cases in 1900, where, among those who attended, several contracted the disease. In India this mode of infection is probably common in the ill-ventilated houses, which are dark, dirty, and densely crowded, and where the atmosphere is breathed over and over again, becoming laden with plague bacilli, and highly infectious to those inhaling it. In well-ventilated houses and hospitals, the atmosphere may be disregarded as a carrier of plague infection. This loss of infectivity of plague in the presence of efficient ventilation resembles closely what is observed in typhus fever. Kitasato claims to have demonstrated the presence of the *bacillus pestis* in dust from the walls of a room occupied by a plague patient. Experimentally, plague can be produced in rats by placing a small quantity of a plague culture on their nasal mucous membranes.

(3) *Ingestion.*—Beyond infection through the tonsil, which may occur in cervical cases, there is no evidence of the ingestion of food causing plague infection. Infected food might conceivably infect wounds on the hands or lips. Childe has stated, as a result of his Indian experiences: "With regard to infection by food, no evidence was found post-mortem that the bacillus had entered through the stomach or intestine." The intestinal type of plague might be cited as a proof of food infection, but it is doubtful whether this type occurs apart from one of the other well-marked forms of plague. Rosenau states that the *bacillus pestis* can live a long time in milk, cheese, and butter, but it usually dies out quickly on the surface of fruits and prepared foods.

In animal experiments it sometimes happens that a healthy rat consumes the body of one of its own species which has succumbed to plague, without itself developing the disease. On the other hand, several observers note that in like circumstances such animals have contracted the disease in a virulent form.

B. Means of Spread.—(1) *Plague in the Lower Animals and Infection to Man. Mammalia—Primates.*—The brown monkey (*Macacus radiatus*), the grey monkey (*Semnopithecus entellus*), and the bonnet monkey (*Macacus sinicus*) are all very susceptible to plague, and instances are reported from India in which monkeys have acquired the disease in nature. They can therefore aid in spreading the disease, though their influence is probably small.

Rodentia.—The grey rat (*Mus decumanus*), the black rat (*Mus ratus*), and the white rat are all naturally susceptible to plague, which may occur epidemically among them. The influence of these animals in diffusing plague will be considered presently. Mice, the pig rat or bandicoot (*Nesokia bandicota*), the grey striped squirrels of India, and possibly porcupines can naturally acquire the disease. Ferrets, used to exterminate rats, frequently contract the disease in plague-infected localities (*e.g.* South Africa).

Outbreaks have been recorded among small rodents of the nature of marmots, locally known as tarbagan (*Arctomys bobac*) in the Transbaikal Province of Eastern Siberia. This disease appears to be transmitted from the marmot to man, and upon the infection of one member of a household several cases in the family frequently occur. There is no doubt that this disease is true plague. It breaks out periodically in the marmots about the autumn, just before the hibernating season, the symptoms observed being unsteadiness in gait, languor, and glandular swellings; the disease is very fatal (Clemow, *Journal of Trop. Med.*, Feb. 1900).

Marsupials.—Wallabies are reported to have become infected in Australia during 1902.

Carnivora.—Cats may acquire the disease, instances being reported from Australia, Mauritius, South Africa, and lately in British East Africa. Suspicious sickness and deaths among these animals have been reported on ships which have had human plague cases on board, sometimes before the manifestation of the disease in man.

Invertebrata.—Bugs, fleas, and flies have been

supposed to act as carriers of plague infection, a subject which is dealt with later.

The Connection between Plague in Rat and Man.—The principal ways in which plague is believed to be spread are by contact directly with persons suffering from the disease or with material infected by such persons or plague-stricken rats.

Those epidemics of which we have fullest particulars naturally divide themselves into two classes, those in which rats have appeared to be the important carriers of infection, and those in which plague-infected rats have been absent or an unimportant factor.

Upon this classification we may draw up the following table, a few outbreaks of both types being selected from the many plague epidemics which have occurred of late years:—

Outbreaks in which Plague Rats have been important factors in spreading Plague.	Outbreaks in which Plague Rats have been unimportant factors.
Sydney, 1900.	Oporto, 1899-1900.
Cape Town, Port Elizabeth, Mossel Bay — 1901-2.	Glasgow, 1900.
	Liverpool, 1901.
Report of Indian Plague Commission: Chak Kalal, Punjab.	
Report of Indian Plague Commission: Mahlgahla, Punjab.	
Report of Indian Plague Commission: Kurachi, 2nd epidemic.	Kurachi, 1st epidemic.
Report of Indian Plague Commission: Poona, later epidemics.	Poona, 1st epidemic.
Report of Indian Plague Commission: Belgaum, 2nd epidemic.	Belgaum, 1st epidemic.
Alexandria, Port Said — 1900.	
	Certain villages in Punjab.

The Plague Commission summarised their views on the connection between plague in rat and man:—

"It has been clearly established that when the infection of plague has been imported into a town or village, rats have, in many cases, contracted the infection before any indigenous case occurred among men. Again, it has been clearly established that, where rats have become infected, they have been instrumental in spreading plague in evacuated villages, and in carrying the infection outward from evacuated quarters. Hardly less clear is it that rats have been frequently at work in disseminating the infection of plague in inhabited towns and villages, and that in some cases they have been responsible for the occurrence of an explosive outbreak of plague among men. It is, however, important to note that the outbreak of plague among rats has not in all cases been followed by the occurrence of plague among men, and that explosive outbreaks have sometimes been due to causes other than the spread of infection by rats."

The following conclusions are therefore arrived at regarding the relative importance of rats and men as agents in disseminating plague in an infected place:—

(*a*) "In some places where severe epidemics have occurred among men, rats have not been infected at all, and in many places where there have been plague epidemics among men, the disease has not appeared among rats in epidemic form.

(*b*) "Occasionally where there has been considerable mortality among rats, there has been an absence of plague among men.

(*c*) "Even when there has been the largest amount of plague among rats, the risk of persons who came in contact with these infected rats appears not to have approached that undergone by persons inhabiting houses[1] in which there have been plague patients, or persons coming into contact with pneumonic plague. There are, however, cases, such for instance as the cases of Mahlgahla and Chak Kalal (already referred to), where a very considerable number of plague cases in a village has been directly attributable to the dissemination of infection consequent on an epidemic of plague among rats.

(*d*) "Since the number of plague bacilli escaping from the infected human organism must be presumed to be greater than the number which escape from a plague-infected rat, *a priori* it would seem clear that a single infected man must constitute a greater danger than a single infected rat; but, on the other hand, seeing that the excreta of rats may be deposited anywhere throughout a house, and that the number of rats which are affected may many times exceed the number of men who are affected, a severe epidemic among rats must, we think, tend considerably to the spread of infection.

(*e*) "The chief importance of rats in the epidemiology of plague seems to us to arise in connection with the first outbreak of the disease in an affected place, for we have seen that in many instances rats become infected before men, and they scatter plague broadcast over an uninfected place.

(*f*) "When plague is once established in a place, we have no doubt that human agency is a more important factor in spreading the disease than the agency of rats." This last conclusion, however, cannot be applied to plague in South Africa, where infection from rat to man appeared to be the common mode of infection.

As to the manner in which the plague rat

[1] It is possible that in many of these houses plague rats were present about the house, under floors, ceilings, etc., or in outhouses.—*J. S. L.*

communicates infection to man, nothing very definite can be stated.

In the minority of cases direct contact with a recently dead rat can be ascertained. Usually only the presence of the plague rat under the floors and ceilings of the house, or among bales and boxes in the house or store, can be proved. In rat-infested places their fæces are found all over the building, proving their wide distribution throughout the house, and we know that the *bacillus pestis* occurs in the urine, fæces, pus from buboes, and bronchial secretion of plague rats. Hence the infection of dust, food, clothing, and anything else in the house is extremely probable; in this way infection may reach man.

The rat flea theory of infection advanced by Simond is an attractive one, but sufficient proof of its truth has not been demonstrated. He holds that fleas from the infected rat become parasitic on man and inoculate him with the disease. In support of this he points out the phlyctenules, which are sometimes found on the same part of the body as the bubo, and which might be produced by a flea-bite. This theory received some support in Sydney, and experiments made there to some extent confirmed Simond's views. Simond's experiments have, however, been adversely criticised by several writers.

Nuttall (*Journal of Trop. Med.*, March 1902) says: "The evidence in favour of the flea-hypothesis is worthless and cannot withstand scientific criticism." He found negative results in a series of experiments, but thought that flies (*Musca domestica*) might be a source of danger in disseminating plague.

Galli-Valerio found that the two varieties of fleas most commonly infesting rats and mice, the *Typhlopsylla musculi* and *Pulex fasciatus*, would not bite him, and readily left him unless confined on his body. The *Pulex erinacei*, which is also found on rats, bit man, but not readily. The common flea of dogs and cats, and sometimes found on rats, the *Pulex serraticeps*, readily bit man. All these fleas can be readily distinguished from the *Pulex irritans* which infests man. Galli-Valerio, therefore, came to the conclusion that *Pulex serraticeps* and *Pulex erinacei* might sometimes convey plague from rat to man, and that *Typhlopsylla musculi* could convey plague from rat to rat. The rat fleas which attack man are, however, either rare on the rat or do not readily bite man (*Journal of Trop. Med.*, 1st Feb. 1902, and *Centralb. f. Bakt.*, Band xxvii. and xxviii., 1900).

I have myself rarely entered a plague-infected house in Cape Town or Port Elizabeth without experiencing several flea-bites, which, however, proved non-infective, and this experience was shared by the numerous disinfecting gangs employed in such houses under my direction. Cases of plague among the disinfecting staff are uncommon, considering their frequent opportunities of becoming infected, but this may be due to their having been, in most cases, inoculated with Haffkine's prophylactic.

To summarise this subject, it seems probable, though the question cannot be considered to be settled, that while fleas may in some cases inoculate the disease or furnish in their bite an aperture through which plague infection may be introduced, the common method in which the rat infects man is by contact of abraded skin surfaces of man with material infected by plague rats.

(2) *Plague Infection from Person to Person.*—It is a recognised fact that plague in a well-ventilated hospital is only infectious to a slight degree.

The only cases which appear to be at all infectious in hospitals are the pneumonic and septicæmic cases. A well-known example of this was seen in the Vienna group of cases in 1898, in which a nurse and a medical attendant both contracted plague pneumonia while in attendance upon a case of this type of the disease. It is to be noted that plague pneumonia tends to reproduce itself, as is well seen in the *Backergunge* outbreak, quoted by the Indian Plague Commission, where twenty cases of this type, extending through six generations of infection, occurred.

Bubonic cases are not as a rule very infectious, the bacillus being for the most part localised in the affected glands and their immediate surroundings, and only entering the blood-stream towards the approach of death, *i.e.* when septicæmia has set in. Such cases might then become infectious by the agency of fleas (if such a mode of spread be accepted), and in addition, such cases are infectious through the lungs, fæces, urine, and hæmorrhages which may occur. As regards the infectivity of the urine, in Sydney, out of experiments in twenty-nine cases, ten showed plague-like bacilli in the urine, but these could not be isolated, and inoculation of animals furnished negative results. Further, plague bacilli failed to grow in human urine, whether sterilised by heat or by passing through a Pasteur-Chamberlain filter, and whether left acid or neutralised. On two occasions, however, growths on agar were obtained from the urine of guinea-pigs which had died of the disease. The bacilli of plague have been stated to have been recovered from the fæces, but most observers state that they have failed to do so. It is possible that the numerous difficulties attending the separation of the organism may account for their failure. It is necessary, therefore, to consider both the urine and fæces to be infectious, especially in septicæmic plague and possibly in other types, though the presence of the bacillus in these excretions has not been satisfactorily determined. The plague bacillus may also gain exit from the body when the bubo is opened, though here again many

observers have failed to recover the bacillus from the pus of a discharging bubo. It has been done, however, and for an unknown length of time the discharging bubo remains a source of infection. In cases with parotid and submaxillary buboes, the *bacillus pestis* has been found in the saliva. In some plague epidemics pulmonary complications of varying degrees of severity occurred in a large number of cases, as in the early part of the 1899 Bombay epidemic. Here, even where the lung changes were slight, plague bacilli were found in the sputum.

This part of the subject may be summarised as follows:—Bubonic cases are at first little infectious, but upon the development of lung symptoms, septicæmia, or the opening of the bubo, become more infectious; primary plague pneumonia and septicæmia are infectious from the beginning, though this infection does not as a rule operate in well-ventilated houses or plague hospitals, in spite of the lack of personal disinfection and precautions on the part of the attendants on the sick. Plague in insanitary, plague-rat infected, or badly ventilated houses becomes a very infectious disease.

(3) *Infected Clothes.*—Clothes can retain their infective property for a long period. Many cases are on record where evacuation of a village or houses has not at once checked the occurrence of plague cases among the inhabitants until their clothing has been removed and disinfected. Another fact bearing on this point is the heavy incidence of plague among the washerman class, who have suffered severely in India. A case of this nature occurred in Glasgow, where the wife of a man employed in collecting infected clothes contracted the disease, the husband, who had received a prophylactic dose of Yersin's serum, escaping infection. There are cases of discharged *employés* from plague hospitals infecting their families at home, probably due to the conveyance of infected clothing which had been hidden to escape steam disinfection.

Bacteriological examination has usually proved negative in the cases of clothes and merchandise, unless grossly contaminated. Infected clothing has played an important part in some cases in infecting sailors, who, upon arrival in port, have opened their boxes, or taken from the bottom of their boxes clothing obtained in a plague-infected port. Such cases occurred in London Docks in 1896, in men who had shipped from Bombay and who had arrived in London in a healthy condition. In some outbreaks, dealers in second-hand clothing, or persons engaged in rag-picking, have been the first attacked. Rosenau states that the *bacillus pestis* can live on clothes for months, when dry in albuminous media at a temperature under 20° C.

(4) *Infected Soil.*—The Indian Plague Commission note that the plague bacillus has not been recovered from the soil by any trustworthy observer in India. There are many difficulties in the way of such a research, and it is posssible that these may yet be overcome. It is probable that the plague bacillus is frequently present on the surface of mud and cow-dung floors, from gross contamination by sputum, excreta, etc., and thus may infect the barefooted inhabitants of the house. As to a saprophytic existence of the bacillus in the soil we know little or nothing, though it has been observed that abundant growths occur in tubes containing plague cultures buried in the soil, possibly from the ample supply of CO_2, which appears to aid the growth of the organism.

(5) *Alleged Infection of Grain.*—When grain is artificially infected with plague, the bacillus soon dies out, and observers have failed to recover the organism after four to six days. In decomposing grain it dies out sooner, the acid produced being fatal to the life of the bacillus. From these experiments it would seem that grain, even if at first directly infected, would soon lose its powers of infection. From the actually infected grain must be distinguished cargoes or accumulations of grain infested by plague rats. In this case the epidemic may be carried on among rats, and on arrival at a port these rodents may infect the docks from communication between the ship and dock rats. The danger, therefore, in grain stores and grain ships does not appear to lie so much in the grain as in the rats with which they are associated. In other words, grain is no more dangerous as a source of infection than any other cargo or store likely to attract rats, and the danger lies in the rats and not in the cargo.

(6) *Water.*—The plague bacillus, according to most observers, readily dies out in water, though Rosenau states that the bacillus can live a long time in this medium. There are no grounds for supposing that plague has ever been a water-borne disease.

(7) *Plague-infected Houses.* — The common occurrence of plague-infected rats in these houses has already been referred to. Regarding the infectivity of the house in which plague has occurred, the Indian Plague Commission observe: "The universal experience of plague in India proves that houses into which the infection of plague has been imported, whether by man or rats, are infective; this infectivity being so marked that many of the officers who have had most experience of the disease have come to the conclusion that the principal source of the infection is . . . to be found in the houses into which the infection of plague has been introduced."

The *bacillus pestis* has not been recovered from the mud or cow-dung floors in India, but this may be explained by the difficulties of the investigation, and not by the absence of the bacillus. Reference has already been made

to the infectivity of ill-ventilated dwellings. In reference to infected premises, Chalmers has stated: "Except for the accidents of exposure to gross and probably prolonged contagion, it is necessary to live the life of the inhabitant in order to incur his perils. And peril exists wherever the surroundings are of a character so grossly insanitary as will afford the bacillus a suitable soil for saprophytic growth."

The influence of insanitary surroundings is also important in this connection, but is referred to in the succeeding section.

(8) *Insanitary Conditions.*—These must be considered as being intimately connected with plague infection for several reasons. In the first place, those living under such conditions have a weakened resistance towards infectious disease in general, and under certain conditions may become directly infected by living in an infected atmosphere vitiated by uncleanliness, insufficiency of ventilation, and overcrowding. This view has been held by Sir T. R. Fraser, the President of the Indian Plague Commission, who regards overcrowding as chiefly operating in adding to the air pollution, and that people living under such influences become predisposed to attack. He holds that the virulence of the plague organism becomes exalted by increase in moisture and CO_2 in the atmosphere, and elevation in temperature, diminution of oxygen, and deficiency of light. The majority of the Plague Commission were unable to accept the view that insanitation had any special influence on plague infection. In the second place, insanitary conditions are almost always associated with the prevalence of rats, and in a rat-borne epidemic these are the places which are naturally the first to become plague infected.

It is necessary, therefore, to regard insanitation always as a predisposing factor, and sometimes as directly connected with the spread of plague infection.

(9) *The Influence of Season and Temperature.*—Season appears to have some influence on the epidemicity of plague, but an epidemic of this disease may occur under any climatic condition. The first Bombay epidemic (1896) attained its maximum at the coolest period of the year, in December; the second reached its highest point as the weather was getting warmer, in February; in the third epidemic it was deferred till the month of March. It is noteworthy that the height of the epidemic has been reached in Bombay city in the month of March in the years 1898, 1899, 1900, and 1901. In all the Bombay city epidemics, the maximum has been reached in the early months of the year, the disease dying down in the hot weather, in May to July. Outbreaks in Calcutta and Kurachi occurred during the hottest season of the year, in Poona during the monsoon, and in the Southern Mahratta country at the close of the monsoon, while winter was coming on. The Cape Town epidemic of 1901 occurred towards the latter part of the hotter weather and during part of the cold weather, when it subsided. A study of the scattered endemic centres of plague shows that the disease can remain endemic in the cold winters of Siberia as well as in the tropical heat of Central Africa.

C. The Spread of Plague from Place to Place

(*a*) By Land; (*b*) By Sea.

(*a*) *By Land.*—Plague may be conveyed to an uninfected locality by infected persons, infected rats, infected clothes, and possibly by infected goods.

Infected rats probably travel but short distances on foot, and spread the infection to neighbouring villages, or to uninfected parts of an infected town. They also travel concealed in bales of goods, and may thus proceed by train long distances. It is probable that though they may die on the journey, the infected carcase may act for a time as a centre of infection to rats or man at its destination, and by handling the polluted goods an outbreak in man may be set up. A case of this nature occurred in Port Elizabeth, where a European had occasion to unpack a bale of goods at his warehouse after it had come from the docks, where many plague rats had been found. A rat, afterwards proved to have died from plague, was found among the goods, and four days after the man was attacked by plague. The only source of infection at all probable was the handling of these infected goods. Had this bale gone up country it is possible it might have furnished the first indigenous case at its destination. It sometimes happens that the first cases in a locality appear associated with the railway goods stores in an uninfected place, as at Bangalore, where the early cases in the city were all associated with *employés* and their families engaged in the goods stores of the Southern Mahratta Railway. The passenger staff and railway passengers were not attacked till later. During the time railway inspection was strictly carried out in all parts of India, a large number of plague cases were detected, in all stages of the disease, from those in the stage of invasion to the moribund and even the dead. I have found them concealed beneath the carriage seat. The importation of a pneumonic case into the confined atmosphere of an Indian home might readily set up an epidemic in an uninfected place. The Indian Plague Commission were of opinion that plague was carried through India from place to place by human agency. According to some the importation of a single case may set up an epidemic among rats in that place, and through them an outbreak among man is started. There is evidence that this does occur some-

times, and at other times the outbreak among rat and man appears to progress side by side. It is noteworthy that on many occasions plague has not been inclined to spread by land; in South Africa the disease has never gained a footing outside the coast towns of Cape Town, Port Elizabeth, and Mossel Bay, and medical inspection has been sufficient to prevent suspected persons travelling inland to infect other parts of the country; a few isolated cases have occurred here and there, but their prompt notification and isolation has prevented the spread of the disease. The small localised outbreaks which have occurred in England and Scotland have also been readily subdued. In India, however, the case is otherwise, and plague has spread by land over most of the Indian Empire.

(*b*) *By Sea.*—The present pandemic prevalence of plague is almost entirely due to its diffusion by sea. In 1894 Southern China and Assyr (Arabia) were the only parts of the world at all likely to infect the rest of the globe. Reference to the geographical distribution of plague, already referred to, for the years since 1894, shows the gradual development of plague in Eastern ports, Assyr at first having little influence. The infection of Bombay in 1896, probably from Chinese ports, was shortly followed by the gradual invasion of a considerable portion of India, and in 1902 the area of infection was still increasing in that country. In 1897 Assyr probably infected Jeddah and Mecca, and from Jeddah, from Bombay, and from the ports of Southern China very many of the countries of the world have now become infected, though in many cases the importation of infection has not resulted in an epidemic at the port into which plague has been introduced. Among the European ports into which plague has been introduced from infected extra-European places may be instanced Marseilles, Genoa, Trieste, Hamburg, Cardiff, London, Plymouth, Hull, Bremen, Naples, Oporto, and Dunkirk, fortunately in most cases without setting up an epidemic.

Plague frequently exhibits a tendency to spread in a country from port to port (Australia and South Africa), or to places upon navigable rivers (Australia and South America). There are, at least, three possible vehicles of spread of plague over sea, viz. infected man, infected rats, and infected goods and clothing.

There appears to be little or no evidence that plague has invaded a country by the aid of infected goods, where evidence of gross contamination by plague rats is absent (a few cases of infection by clothing have been already noted). On the other hand, there is a good deal of evidence that plague rats have carried infection from country to country. The source of infection of Asunçion (Paraguay) is ascribed to this agency. Rice-bags from India, it is alleged, were transhipped into a sailing ship, the *Zeir*, in which dead rats were afterwards found, and on board which cases of suspicious illness occurred. From the *Zeir* the rice-bags were transhipped into a steamer, the *Centauro*, which called at various Argentine ports on her voyage up the River Plate, and on board which three suspicious deaths occurred among the crew, and on which excessive mortality among rats was observed. A fortnight after the arrival of this ship in Asunçion a large rat epidemic was noticed on the Customs House premises, bacteriological examination confirming the diagnosis of plague. An outbreak of plague in man followed.

The infection of the South African ports is ascribed, as has been said, to infected rats in South American forage which was conveyed in great quantity during the war; the dock *employés* were the first to contract the disease, under conditions already described. Plague in Sydney is supposed to have been introduced from some infected port, the dock rats becoming first affected, and subsequently the town and its inhabitants. Plague rats were also found in the docks at Adelaide, Brisbane, and Melbourne. It is noteworthy that in most plague-infected ports it is the dock *employés* who are first attacked, which would seem to absolve ships' crews and passengers from being the usual carriers of infection, though sometimes cases are detected among sailors at the routine medical examination of the vessel on arrival. The infection seems, therefore, to be conveyed by the ship by some other means than the crew and passengers, and that, it is surmised, is by infected rats and infected goods. We have noted that most of the evidence at our disposal points to infected rats as being the great carriers of infection from country to country. This view is further strengthened by the very frequent association of plague rats in the docks of plague-infected ports; in addition to the Australian and South African ports already referred to, we may instance Alexandria, Port Said, Santos, Rio de Janeiro, San Nicholas, Asunçion, and other South American towns.

While attaching great importance to the importation of the infection of plague by means of the rat, the agency of man as a carrier of infection must not be overlooked. Many instances of ships having human plague cases on board have been detected at their port of arrival. To consider only the cases which have arrived in this country:—In 1896 three cases occurred in London Docks among the crews of steamers from Bombay some days after arrival in port. In 1897 and 1898 two cases occurred in each year during the homeward voyage from India. In 1899 four ships were reported as infected. In 1900 twenty-three ships arrived in English ports with suspicious cases, among whom only four were proved bacteriologically to be plague. In 1901 thirty-three suspected

ships arrived, one ship from Alexandria furnishing nine cases of plague (including two infected while upon attendance on the sick in port), another having had cases on the voyage from South America, and another one case from Cape Town, and two others had had cases *en route* from India. In addition a vessel from Smyrna arrived at Bristol with plague-infected rats. During 1902 only eleven ships suspected of being infected by plague arrived, one with a case on board, other four having had cases during the voyage. Four ships had had a rat mortality on board. Only one of these rat epidemics was suspicious of plague, but the bacillus did not react to all the tests for the specific micro-organism of plague. There was, however, a certain amount of similarity between these bacilli. No cases of plague have occurred in Great Britain beyond ship-borne cases, except the Glasgow cases of 1900 and 1901, Liverpool 1901, and two cases (one of which was not bacteriologically confirmed) at Cardiff in the same year, both employed in grain stores in the docks, where there was a rat epidemic. It is to be noted that all these places are ports, and the employment of some of the household of the first cases in Glasgow and Liverpool, as has been said, has been traced to the docks, but no source of original infection has been determined.

It is obvious that medical inspection of shipping from infected ports is of great value in keeping plague cases out of the country, and in this we have been very successful; but it would appear, in the light of the experience of other countries, that the infection of the ports of this country may possibly occur by means of plague rats rather than by human agency, and a very much more difficult cause to prevent.

5. Symptomatology and Types

Plague may be divided into the following clinical types:—(1) Bubonic plague; (2) Septicæmic plague; (3) Pneumonic plague; (4) Intestinal plague; (5) *Pestis ambulans*; (6) *Pestis minor*.

It must be recognised that these types are not distinct, and that a patient suffering from bubonic plague may be also suffering from plague septicæmia and pneumonia, *i.e.* cases of a mixed type are not uncommon. The essential characteristic of plague is inflammation of the lymph glands, usually those in the groin, axilla, or neck; it is to these cases the term bubonic plague is applied. In the septicæmic type, the internal lymph glands throughout the body, such as the mesenteric and retroperitoneal glands, are found to be affected. In the pneumonic type the glands at the root of the lung are frequently implicated. It is doubtful whether there is a distinct intestinal type of plague. *Pestis ambulans* is a mild form of the bubonic type. *Pestis minor* is a mild glandular complaint which may have some relation to plague, as yet ill-determined.

(1) Bubonic Plague.—After an incubative period, commonly from two to five days, the disease sets in with sudden high fever, preceded sometimes by rigors, sometimes in children with convulsions, and sometimes by prodromal symptoms, as headache, chilliness, nausea, vomiting, vague pains in the back, groins, or axilla. The patient rapidly becomes markedly ill.

Examination of the case in its early stages shows great prostration, high fever, swelling or tenderness in the groin, axilla, or neck, giddiness, loss of appetite, nausea, sometimes vomiting and diarrhœa, pains in the head, back, and limbs, restlessness, sleeplessness, and it may be delirium, together with inco-ordination of speech and gait. Injection of the conjunctiva is frequently well marked; the face is at first flushed, becoming pale later, and the expression may be anxious or apathetic. The pulse is rapid, and the respirations may be increased. The tongue is usually coated, with red edges. The signs and symptoms of plague, as they affect the various systems of the body, are as follows:—

Lymphatic System.—In this system we find the characteristic plague lesions, buboes being present in about three-quarters of the cases, in some epidemics a greater proportion, though in others few of the cases may show enlarged glands. In a short time, varying from a few hours to two days, the characteristic bubo makes its appearance. There is first a mere tenderness in the groin, axilla, or neck—it may be only upon pressure. Then a slight fulness and redness appears, with increasing tenderness. It is not uncommon to find one or more enlarged glands quite distinct from one another. Later, with periglandular effusion, they become agglutinated together. The term bubo does not mean an inflamed gland, but one or more glands matted together, along with the periglandular infiltration.

These changes may succeed one another with great rapidity, a surprising difference being manifested in but a few hours. The swelling may reach the size of an orange, or a man's fist, or even larger. Pain on palpation, as a rule, is exquisite; the neighbourhood of the glands implicated reveals a considerable amount of œdema, so that the gland itself can be made out sometimes only on deep pressure. The patient often lies with a flexed limb to relieve pressure on the bubo, his attitude giving sometimes a hint as to the site of the glands affected. Deep palpation in the iliac region will often reveal the presence of inflamed glands, which are especially associated with inguinal and femoral cases; occasionally these iliac glands may be the only ones implicated, and may give rise to some difficulty in diagnosis, as sometimes they cause little pain till they are palpated.

The epitrochlear and popliteal glands are occasionally affected, but nearly always in association with buboes elsewhere. As has already been stated, the groin is the commonest site for the bubo, then come in order of frequency the axilla, neck (cervical, parotid, and submaxillary glands), epitrochlear and popliteal glands. This probably depends on the mode of entry of the disease into the body, *i.e.* through abraded skin surfaces, for there is a relation between the extent of the area of skin surface draining into the various sets of glands and the frequency with which these glands are affected, as has already been described. Frequently more than one region of the body shows buboes, and in each region affected there is usually more than one gland implicated.

In this disease some part or other of the lymphatic system is always affected, though often this cannot be discovered until a post-mortem examination has been made. In the bubonic type the bubo is always evident, though in the rarer cases of pelvic buboes the affected glands may be overlooked unless due care be taken.

The œdema which is always present in the periglandular tissues may extend over a wide area, causing considerable swelling over the thighs and lower part of the abdomen or over the sternum and shoulder blades. The trachea and larynx may be pressed upon, causing dyspnœa. Hæmorrhages may discolour the skin over a considerable area.

As the degree of coma of the patient increases, the pain produced on palpation may not be so apparent, though, even where considerable coma is present, palpation of the bubo will produce evident pain and expostulation, or it may be merely a groan.

If the patient lives, the buboes resolve or suppurate, and this is frequently very prolonged. Suppuration usually occurs in the second week of the disease; sometimes it may be delayed for a considerable time. The whole gland or glands and surrounding tissues may slough *en masse*, leaving a large deep ulcer, which finally granulates up. Often a very obstinate sinus persists for a long time. Cases sometimes occur where gland after gland in different parts of the body becomes involved and breaks down, the process going on for several months, and occasionally cases occur where glands break down after discharge from hospital; these are rare, but one or two such cases occurred in Cape Town during the recent epidemic.

The temperature has sometimes by the second day attained its maximum; at other times it rises gradually till the third or fourth day, when it frequently falls a little, and in mild cases it begins to descend to the normal. Generally, however, the temperature rises again till the fifth or seventh day, when it descends by lysis to normal. This may be followed by an intermittent fever caused by septic absorption. A fresh elevation in temperature may occur from fresh glands becoming involved, or from secondary pneumonia. Frequently before death the temperature may attain a great height, such as 108° F. or even higher, and continue rising after death to over 110° F. when taken in the rectum. Sometimes the temperature just before death is subnormal.

Respiratory System.—The respirations are increased, being usually thirty to forty per minute. Besides plague pneumonia, which may be primary or secondary, and which is described later, bronchitic symptoms and œdema of the lung may supervene. Lung complications in the bubonic type vary in frequency in the different epidemics.

Circulatory System.—In the early stages of the disease the pulse is full and dicrotic. Even in very mild cases the rapidity of the pulse is increased and may afford considerable diagnostic assistance in abnormal cases. It may also give information that the patient's condition is more serious than would otherwise be imagined. Patients very ill, and sometimes within a few hours of death, may be able to walk about; examination of the pulse at once reveals their critical condition.

The heart may become dilated, the apex beat may be diffused, and murmurs may be audible at the apex and in the pulmonary area. There is great tendency to heart failure in this disease, and it may come on very suddenly without any warning. There is generally leucocytosis and sometimes diminution in the number of red blood corpuscles.

Alimentary System.—The tongue is usually coated at first and has an appearance resembling chalk or mother-of-pearl; the edges are clean and red. Later the tongue becomes dry and cracked and of a brown colour. It is not uncommon for patients to die with quite a moist tongue. The teeth, lips, and nostrils become crusted, and the lips dry and shrivelled. Nausea and vomiting are common at the onset and for the first day or two of the disease. Constipation is common at the beginning, followed by diarrhœa, the stools sometimes containing blood. There may be abdominal tenderness and distension. Thirst is usually a marked symptom.

The liver and spleen are enlarged and are usually tender, particularly the spleen.

The Urinary System.—The secretion of urine is diminished and may be suppressed, associated with lumbar pains. Retention of urine is not uncommon, and the catheter may have to be employed. The urine is of a dark colour, strongly acid, high specific gravity, and contains urates and sometimes true and nucleo-albumin. Microscopically, granular, hyaline, and rarely fatty tube casts may be found, together with

blood corpuscles. The Austrian Plague Commission found that the chlorides were in almost all cases markedly diminished.

The Nervous System.—This system is especially implicated in plague. Every degree of mental disturbance may be met with; apathy, anxiety, delirium of varying degrees, or acute mania may be present, and suicidal and homicidal impulses may supervene. The mind is nearly always more or less affected; sometimes it is merely clouded, or memory is defective and the attention frequently wanders; sleeplessness is a troublesome symptom. Inco-ordination of speech is usually well marked, the patient often knowing what he wants to say, but has difficulty in articulating it. Inco-ordination of the muscles is often well marked; twitching, picking at the bed-clothes, tonic and clonic spasms may be present. The speech is usually thick and the gait staggering, and these, together with the conjunctival injection, may present a picture suggestive of alcoholic excess.

At times, even in cases where death is imminent, the mind is but little affected and the patient painfully realises his critical condition. Restlessness and purposeless movements are of frequent occurrence among those attacked by plague, leading them to make train journeys and expeditions seemingly without an object; a part of this, however, is explicable, especially in native races, by the fear of being discovered as a plague patient, or to the promptings of friends. Nervous sequelæ may follow plague, such as motor paralysis and insanity.

Cutaneous System.—The skin during the high fever is hot and dry, followed by perspiration on the fall in temperature. Primary and secondary plague boils, pustules, and carbuncles may occur. The primary lesions occur at the point of inoculation of the disease, beginning as a small papule and going on to pustulation. The *bacillus pestis* may sometimes be recovered from the pustule in nearly pure culture. There is usually considerable surrounding redness, and it may be lymphangitis between the pustule and the nearest lymph gland; vesicles, pustules, and furuncles may develop along this track. Occasionally a type of plague has been described, limited to the skin, under the name "Skin Plague or Pustular Plague." The general symptoms resemble bubonic plague, but are milder, and the termination, apart from the local skin destruction, is more frequently favourable; the carbuncles usually take an abortive course and dry up, unless other local or general symptoms appear (Scheube).

Secondary plague pustules, boils, and carbuncles are sometimes met with, usually during the first week of the disease. Accompanied by a feeling of heat, stinging or itching pains, there appears a small brown spot over or round a bubo, surrounded by redness and induration. A small vesicle develops with a dark red margin, which becomes larger, and may reach the size of a Brazil nut. The base of the vesicle becomes a black slough, which in time becomes a black ulcer of crater-like formation. In process of time the ulcer heals by granulation (Scheube). Similar sloughs may be produced by native applications to the bubo, as occurs in India. Sometimes more extensive carbuncle formation occurs with necrosis and loss of tissue, so that a large extent of muscle may be laid bare, and sometimes hæmorrhages may supervene.

Petechiæ may be present, commonly on the neck, chest, and extremities. They may occur as small purpuric spots or partly punctiform and partly wheal-like, or large red, black, or livid hæmorrhages. The incidence of such cutaneous affections appears to vary very much in different epidemics. Papular rashes are sometimes met with, distributed all over the body. Sometimes these are said to go on to pustulation. Rashes have from time to time been described, which have a typhus-like, morbilliform, erythematous, urticarial, or sudaminous character, but they are rare. Typhus-like rashes have been described by Hossack, in Calcutta (*Journal Trop. Med.*, Ist January 1901), though in his cases drugs may have had something to do with the causation of the rash.

Hæmorrhages.—Bleeding may occur from the nose, mouth, lungs, stomach, rectum, kidneys, and female genital organs, usually at the height of the disease. Abortion and premature labour frequently occur, and result almost invariably in death; cases of recovery are, however, upon record.

Eye affections may occur early, even on the first or second day of the disease, such as parenchymatous keratitis of one or both eyes, leading to iridocyclitis usually, and sometimes to complete suppuration of the eyes.

Termination.—Death may occur at any stage of the disease, but most frequently on the third to fifth day, so that after a week the prognosis becomes more favourable. There are well-marked fulminant cases (*pestis fulminans* or *siderans*) where the patient has been able to attend to his work most of the day apparently in good health, and on going home has been attacked and died of plague the same evening. Although it is true that Europeans, as a rule, do not suffer so severely from plague, I have seen some cases of this fulminant type among them; in some instances the disease ran its course in six or eight hours. In view of such cases, sudden deaths during a plague epidemic should be regarded with suspicion until the diagnosis can be satisfactorily cleared up.

In plague the cause of death is usually heart failure, which may be very sudden and unexpected, or ushered in with a small, frequent, weak, and irregular pulse, coldness of the peripheral parts of the body, high temperature, and sometimes cyanosis or lividity of the face.

Other causes of death may be asphyxia from extensive inflammation and infiltration in the neighbourhood of the throat, or hæmorrhages from the lungs, or from ulceration into large blood-vessels elsewhere. Death may occur from meningitis, pyæmia, septicæmia, pneumonia, or marasmus. Patients have been known to become maniacal, and to have died violent deaths by jumping through windows, or on board ship through portholes into the sea, and in other like ways. In favourable cases the disease passes on slowly to convalescence, through remission of the severer symptoms, between the first and second weeks. Progress may be slow, and the incised bubo may sometimes take a long time to heal. Some considerable time may elapse before the patient regains his normal health. Cases suffering slightly from pulmonary tuberculosis may rapidly die from an acute form of the disease. Bubonic cases which have not suppurated will, in general, require to remain in hospital from five to eight weeks. In those in which suppuration has occurred the incised bubo, in a number of cases, heals fairly readily, and complete convalescence may not be unduly protracted. In some cases three or four months may occasionally be necessary before a firm cicatrix is established or before glands in different parts of the body cease to break down. It is necessary to consider a gland infectious as long as it is discharging, though probably after some weeks it no longer contains the specific organisms.

(2) Septicæmic Plague.—This form of plague consists of rapidly fatal septicæmia, terminating in a few hours or a day or two in primary cases. Secondary septicæmia or pyæmia may supervene in bubonic cases, and are common causes of death. For statistical purposes, however, these secondary cases are not regarded as cases of septicæmic plague, but are classified as bubonic. In this connection it may be noted that although it is convenient for purposes of description and classification to divide plague into several types, in practice it is common to meet with mixed forms of the disease.

The term septicæmic plague may therefore be confined to a primary septicæmia, without external buboes. The onset is frequently sudden, prostration is extreme, the patient may lie in a collapsed or comatose condition, he may be delirious, and sometimes breathes with a groaning respiration, or only a groan may be elicited on interrogation. On examination a weak, irregular pulse, and sometimes an enlarged spleen and hæmorrhages from the mouth, rectum, or female genital organs may be discovered; and in females it may be found that the patient has aborted or been prematurely delivered, the so-called "puerperal" form of plague. The temperature is usually hyperpyrexic, or it may be apyrexic. Without a bacteriological examination diagnosis is uncertain, but in blood taken from the peripheral circulation the specific bacillus can often be discovered in films or upon culture.

Pestis fulminans and *siderans* belong to this type of the disease. The termination is fatal in practically all cases.

(3) Pneumonic Plague.—Plague pneumonia may be primary or it may be secondary to an attack of a bubonic type.

Childe first noted this primary plague pneumonia in Bombay, though plague epidemics with lung symptoms are mentioned in some of the early writings upon this disease. Commencing with rigors, high temperature, headache, pains in the limbs, and giddiness, pneumonia develops with pains in the chest, dyspnœa, cough, expectoration, and accelerated breathing.

The physical signs are often obscure and evanescent. There may be dulness over one or more lobes, or it may not be definitely made out. Moist sounds are usually heard, and occasionally bronchitic râles. The fever is high, intermittent, and often irregular, the pulse frequent and weak. Delirium is a marked feature of plague pneumonia, passing on to coma as death approaches. As points in the differential diagnosis of this form of the disease, the prostration is very marked and out of proportion to the physical signs. The pulse and respirations are rapid, but there is not the same disproportion in their ratio as is observed in ordinary pneumonia. There may be tenderness in the groins, axilla, or neck, and frequently enlargement of the spleen. It is stated that *herpes labialis* is rarely or never present.

In the early stages the sputum may be thick and tenacious, and slightly tinged with blood; it later becomes a watery, blood-stained secretion, sometimes consisting of almost pure blood; it wells up in enormous quantities and is generally laden with plague bacilli, sometimes in nearly pure culture. The rusty sputum of ordinary pneumonia is seldom met with in this form of the disease. The Russian Plague Commission state that cough and expectoration may be entirely absent.

The disease has in the great majority of cases a fatal termination in three or four days from œdema of the lung. Sometimes the disease takes the form of what seems to be a simple bronchitis, suddenly terminating in death. In certain epidemics, cases of plague pneumonia are not reported to have occurred. In the 1894 outbreak in Hong-Kong, this type was either not present or, what is more likely, was not recognised, Childe at that time not having described this form of the disease. Sometimes an outbreak consists almost entirely of pneumonic cases.

(4) Intestinal Plague. — It is doubtful whether this should be considered to be a distinct type of plague, but it was described by Wilm in Hong-Kong, 1896. In it no buboes

occurred, but all the symptoms were referable to the intestinal tract, with vomiting, diarrhœa, and passage of bloody slime per rectum. It has not been observed in Bombay. Personally I have only observed intestinal symptoms in those cases which have also been suffering from one of the other well-marked types of plague.

(5) Pestis Ambulans.—This term should be reserved for those cases of true plague in which the symptoms and constitutional disturbances are mild. There may be pain in several groups of glands and slight fulness or swellings in the groin, axilla, or neck, which swellings ultimately resolve or suppurate. There is some prostration, a varying amount of fever, some conjunctival injection, and may be some inco-ordination of speech; the tongue has a creamy coat with clean edges. The characteristics of this form of the disease are therefore those of a mild bubonic type of plague, not readily recognised unless looked for with care, and apt to be mistaken for mumps, syphilis, tubercular glands, or the results of strains and injuries. It is stated that such ambulatory cases occasionally terminate suddenly and unexpectedly in death, though I have not met with such instances.

In chronic cases of this type suppuration may be prolonged. These ambulant cases are regarded by many authorities as being a great source of danger to the community by going about with impunity spreading the disease. There is not much evidence in favour of this view.

(6) Pestis Minor. — This term has been applied to a mild epidemic disease characterised by some fever and the formation of buboes, usually in the groin, which after a time resolve or suppurate. In some cases epidemics of this kind have preceded by a few months or years epidemics of true plague, such as happened in Mesopotamia (1876-77), Astrakhan (1877), Singapore and Straits Settlements, and the south coast of China (1893). *Pestis minor* was also reported to be present in Calcutta in 1896. Epidemics of this disease have been observed without true plague following them, and true plague epidemics are usually, as far as is known, not preceded by outbreaks of *pestis minor*. It is at present uncertain whether such epidemics are due to the *bacillus pestis*, a question which can only be cleared up by bacteriological investigation. There is no doubt that there are epidemics or groups of cases of non-venereal glandular disease, but they may be due to several causes, some perhaps being due to the plague bacillus. In these glandular fevers, or so-called "climatic buboes," the glands and skin over them often break down. The term *pestis minor* has been misapplied to *pestis ambulans*, but it is better to keep them distinct.

Sequelæ.—In addition to long-continued weakness which may follow an attack of plague, rendering mental and physical exertion an impossibility for some time, the following sequelæ may be mentioned:—Long-continued suppuration of glands, boils, and carbuncles, joint inflammations, especially the knee joints, paralysis of various kinds (face, muscles of the eye, palate, larynx, etc., hemiplegia), ataxia, mental disturbances, loss of speech, deafness in one or both ears, or the sight may be affected by intraocular changes; *i.e.* the commonest sequelæ are nervous or suppurative.

Incubative Period.—The number of cases in which the incubative period can be definitely settled is small, but such as they are seem sufficiently conclusive. The Indian Plague Commission found in 13 cases of accidental inoculation, in the course of post-mortem work, the incubative period was from one to three days, and in 16 cases in which the occasion of coming into contact with an infected person was known the period was from eight hours to five days; the average period in both series was under three days. This coincides with the experience of those who have been much engaged on plague work, the incubative period rarely exceeding five days. Perhaps cases of prolonged incubation do occur, but it is often difficult to entirely eliminate other sources of infection in such cases. For practical purposes seven days can be regarded as the outside limit in nearly all cases, and two to four days as the incubative period in the great majority of instances. Preventive measures framed on a fourteen or even twelve days' observation of "contacts" appear to impose needless restrictions.

Mortality.—In many outbreaks it is impossible to arrive at a true mortality of the epidemic on account of the concealment of cases generally practised among native races. In some parts of India the plague mortality has been described as the difference between the actual mortality and the average mortality of non-epidemic years, a method which no doubt, after making certain deductions, gives a truer idea of the plague mortality than the bare record of the reported deaths from this disease. Similarly, among native races it is difficult to get an idea of the true case mortality of a plague epidemic, as no doubt many mild cases escape recognition and many plague deaths are ascribed to other causes. For this reason the appalling case mortality in Hong-Kong may not represent the true facts of the case, as a very large number of the cases were dead when discovered. From 1898 to the middle of 1901 the percentages of dead bodies found among the total number of cases reported were respectively 36, 40, 37·1, and 21·7 per cent. During the first three years of plague in India, from 1896 to 1899, the Indian Plague Commission estimate the mortality at 430,500. In some villages in Dharwar the number of plague

deaths ranged from 22 to 25 per cent of the entire population, a severity paralleled by that of the Black Death of the fourteenth century. There is no doubt that certain races suffer more severely during an epidemic of this disease, both as regards susceptibility to attack and case mortality, partly due to their insanitary surroundings and consequent weakened resisting powers, also to their labouring in infected localities, native habits of life, and lack of early hospital treatment.

In modern plague epidemics of any magnitude, the hospital-treated European cases always show a more favourable result than native cases treated under similar conditions. In Sydney (1900), out of 293 European cases the case mortality was 32·4 per cent, and out of 10 Chinese 8 died. In South Africa (1901 to the early part of 1902), among 221 Europeans 33·9 per cent died, and among 450 coloured people (those of partly European blood, more or less remote) 56·7 per cent died. Among 205 natives (including Africans, Malays, Chinese, and Indians) the death-rate was 44·4 per cent.

The case mortality varies also with the different phases of the epidemic. In the 293 Sydney European cases, we have the following figures :—

20th January to 12th April 1900, 100 European cases, fatality 37 per cent.

12th April to 9th May 1900, 100 European cases, fatality 37 per cent.

9th May to 9th August 1900, 93 European cases, fatality 23 per cent.

In Sydney the disease was at its full virulence from the beginning, and became milder towards the close of the epidemic.

The Indian Plague Commission also found a variation in mortality with the phase of the epidemic. A study of the curve of incidence and curve of case mortality in the case of Bombay city shows that at the beginning and end of an epidemic the number of cases and the case mortality of these are lowest, both curves rising together to a maximum and falling again. In Bombay Presidency and native states connected with it, from September 1896 to 1st January 1900 there were 392,285 *reported* cases, with 308,140 *reported* deaths (excluding Europeans), a case mortality of 78·53 per cent, varying from 83·7 to 59·3 per cent during this period. The figures are certainly very inaccurate, as all statistics must be from countries like India and China, and probably do not give a true idea of the case mortality.

The influence of sex on mortality does not appear to be marked in India or South Africa. Out of 5294 Indian cases collected by the Commission the male case mortality was 71·15 per cent, the female 69·37 per cent. In South Africa, out of 638 males 47 per cent died, and out of 238 females 50·8 per cent. The fatal character of plague in pregnant women has an influence in this connection.

6. Morbid Anatomy and Bacteriology

(1) *Morbid Anatomy.* — The pathological features of plague are congestion of the organs, tendency to hæmorrhages, inflammation and surrounding œdema of the lymphatic glands, with the occurrence of the *bacillus pestis* in the glands and usually also in the internal organs.

(*a*) *External Appearances.*—The body is usually well nourished and *rigor mortis* may be well marked. Examination of the groin, axilla, or neck may show buboes surrounded by considerable œdema. Petechiæ are frequently observed on the chest and limbs. Phlyctenules may be present on the limb upon which the bubo is present.

(*b*) *Internal Appearances—Lymphatic System.* —The entire lymphatic glandular system of the body is frequently involved, even in those cases which show no external bubo. On section of the skin overlying a bubo, a copious infiltration of an œdematous jelly-like fluid and hæmorrhages are met with in the periglandular tissues. The glands on section appear dark red or blackish in colour, very juicy, often soft and pulpy; abscess formation or necrosis of the gland tissue may be found. The bubo may consist of several glands more or less matted together. Chains of glands similarly affected may be found leading from the bubo, and frequently such chains are found leading from the groin to the sacral and lumbar glands, and from the axilla to the mediastinal glands. These secondarily affected glands may show less change than the original bubo. The glands and periglandular infiltration are found to be swarming with plague bacilli.

In pneumonic plague it is common to find the bronchial glands involved, and in septicæmic plague the lymphatic glands in various parts of the body are found to be implicated.

Hæmorrhages are a characteristic feature in the pathology of this disease. They may occur all over the body; in addition to the lymph glands already referred to, they may occur on the surface and in the substance of most of the organs of the body, the commonest sites being the lungs, heart, liver, spleen, and kidneys. The colour varies from a bright red to a reddish black or black, and the size from a minute pinpoint to several inches in extent. In pneumonic plague the lungs nearly always show these hæmorrhages, extending inwards from the surface. Hæmorrhages may also be found in the skin.

Brain, Meninges, and Spinal Cord.—These are usually congested and may show hæmorrhages. A serous or sero-purulent exudate laden with plague bacilli may be found on the pia mater, especially along the vessels, and the ventricular fluid may be increased. The substance of the brain, spinal cord, and nerves present a normal appearance.

Lungs and Pleura.—The pleura may show engorgement, and hæmorrhages and exudates,

clear or blood-stained, may occur in the pleural cavity.

The lungs are generally congested and œdematous, especially the lower lobes. Hæmorrhages and hæmorrhagic infarcts are common, and the thoracic glands may be involved.

In plague pneumonia the typical appearance is that of a confluent lobular pneumonia with scattered irregular patches of varying size from a bean to an egg, grey or red in colour, surrounded by dark red hæmorrhagic areas and œdema. Pleurisy may be present over the surface of these patches. The pneumonic patches may become necrotic. In some post-mortems all one sees may be œdema, and no patches may be made out. In others a whole lobe may be consolidated. The bronchial tubes contain frothy sero-sanguineous fluid, and their mucous membrane may be congested. Crowds of plague bacilli are found in smears from the pneumonic patches and infiltration. The bronchial glands are commonly involved.

The heart is usually dilated, especially the right ventricle. The heart muscle is frequently pale, and may show parenchymatous, cloudy, or fatty degeneration. The visceral layer of the pericardium often shows hæmorrhages of various sizes, especially between the auricles and ventricles. Subendocardial hæmorrhages may be found, and sometimes there are hæmorrhages into the muscle substance itself.

Peritoneum.—Subperitoneal hæmorrhages are common beneath the parietal layer, and upon most of the viscera. Retroperitoneal extravasation of blood associated with inflamed retroperitoneal glands may be found. Extensive extravasations of blood may be found in the omentum and mesentery, particularly in septicæmic plague.

The liver is usually large and hyperæmic. It may be pale and soft, showing parenchymatous degeneration. Necrotic abscess-like foci may be found surrounded by a hæmorrhagic area. Under the capsule of the liver hæmorrhages are frequent, and some authorities state that hæmorrhages on the gall-bladder are very common. Cholecystitis and pericholecystitis have been noted.

The spleen is always enlarged and congested, the colour of its substance being usually very dark, and it is generally soft and is readily broken down. Hæmorrhages under its capsule are of frequent occurrence.

Stomach and intestines frequently show petechiæ and ecchymoses in their mucous membranes. The solitary follicles and Peyer's patches are often swollen.

The kidneys are often swollen and congested, the cortical substance enlarged, and the parenchyma shows cloudy or fatty degeneration. Hæmorrhages, subcapsular or in the pelvis of the kidney, may be seen, or there may be hæmorrhagic infiltration round the organ.

The bladder may contain bloody urine, and its mucous membrane may be ecchymosed.

The uterus and Fallopian tubes may show hæmorrhages in their mucous membranes. The uterus in cases of plague in pregnant women will usually show signs of recent abortion or delivery. These organs should be examined in cases of sudden death in women without a satisfactory medical history.

Post-mortem Appearances in Septicæmic Plague.—The changes are as just described in the internal organs of the body. Hæmorrhages into these organs, and inflammation of the lymphatic glands in the thorax and abdomen, are commonly met with. From nearly every organ the *bacillus pestis* may be recovered.

(2) *Bacteriology.*—The plague bacillus was discovered by Yersin in 1894 during the Hong-Kong outbreak. A bacillus discovered by Kitasato in the same epidemic has been supposed to be identical with that of Yersin, but Kitasato himself has now admitted that Yersin's bacillus is the specific organism of this disease (Report on Plague, *Journal Bacteriologicus Japonis*, December 1899). It is a short oval bacillus with round or square-cut ends, occurring singly or in dumb-bells, and occasionally in chains. It is sometimes surrounded by a capsule. In fresh specimens it frequently occurs in pairs as a diplo-bacillus. It is not motile, nor does it form spores. Its size ranges from 1·5 to 2 μ, and it is ·7 μ in breadth, as average measurements, but it may be longer or shorter than this, and its size is indeed very variable. Mervyn Gordon and others have succeeded in demonstrating the existence of flagellæ.

The bacillus stains easily with any of the ordinary aniline dyes, Löffler's methylene blue (concentrated alcoholic solution of methylene blue, 30 c.c.; potassium hydrate solution (1 in 10,000), 100 c.c.), or very dilute carbol fuchsin being convenient stains. It is decolourised by Gram's method. The bacillus in fresh specimens generally shows a marked tendency, though not always, to a *bipolar stain*, the ends taking up the dye more readily than the middle. Neisser's double stain (glacial acetic acid, methylene blue, and Bismarck brown) also causes this bipolar arrangement to be exhibited. In actively growing cultures the polar stain is nearly always absent.

The bacillus may be conveniently demonstrated from post-mortem specimens or from punctures of suspected glands by making cover-glass preparations, drying, fixing with a mixture of equal parts of ether and absolute alcohol, and staining for a few minutes with methylene blue. After washing, drying, and mounting, the specimen may be examined, and the above appearances observed. In chronic cases, and in those in which the bubo has been present a

few weeks, very few or no plague bacilli may be visible in such films.

The organism is aerobic, but it can grow, though less readily, under anaerobic conditions. In cultures the organism grows readily on all the ordinary laboratory media, forming white, moist, transparent, and iridescent colonies on blood serum at 37° C. in twenty-four to forty-eight hours. Upon agar the colonies are small and round, almost translucent by transmitted and white by reflected light; on contact with a platinum needle they exhibit a characteristic, tenacious, slimy quality, and are not very firmly connected with the culture medium (German Plague Commission). In stab cultures in gelatine it grows slowly along the track of the needle, and the medium is not liquefied. A rapid greyish-white surface growth occurs on glycerine agar. A characteristic growth occurs in bouillon, a white granular deposit growing at the bottom and sides of the tubes, the culture fluid appearing clear. The characteristic "*stalactite*" *growth* occurs in faintly alkaline or neutral peptone broth, to which a few drops of olive, cocoanut, or linseed oil or *ghee* have been added, stalactites of growth hanging from the floating oil-drops (Haffkine). The flask must be kept absolutely at rest at a temperature of 25° to 30° C., when after several days this phenomenon gradually takes place. This is the method made use of in the preparation of Haffkine's prophylactic. After several sub-cultures this appearance may cease to be produced, but passage through a susceptible animal will restore it, or it may spontaneously reappear (Klein).

In sugar-containing media there is no fermentation or gas production.

In old cultures upon agar and bouillon, involution forms of the organism are found, which show great variety in shape, sometimes a long slender bacillus, segmented and vacuolated, at other times swollen and pear-shaped. These forms stain badly. On agar, to which 2·5 to 3·5 per cent salt solution has been added, Hankin and Lcumann found these involution forms appeared in twenty-four hours at 37° C.

Viability of the Plague Bacillus.—The *bacillus pestis* is killed by heating for ten minutes at 62° to 65° C. It loses its virulence by drying, and requires both warmth and moisture for the maintenance of its virulence. The bacillus can, however, grow on media at a low temperature for a long period.

Corrosive sublimate, 1 in 1000, kills the organisms instantaneously; carbolic acid and lysol, 1 per cent, in ten minutes; sulphuric acid, 1 in 1000, in five minutes.

The German Plague Commission stated that the longest time the bacilli in infected material, such as lint, wadding, etc., remained active was eight days. Sputum from pneumonic cases, kept in a vessel plugged with cotton-wool, was no longer virulent after sixteen days. In ordinary drinking water the bacilli die in three days, in sterilised water in eight days, and in sterilised bilge water in five days. In direct sunlight the organism dies in three or four hours. Different observers have, however, arrived at very different results in regard to the action of heat, cold, disinfectants, etc., upon the plague bacillus, dependent partly upon the different climatic conditions under which they were working, and partly also upon the varying degrees of virulence of the bacillus.

The Virulence of the Organism.—This varies in culture and in nature very much, being rapidly lost on artificial media. Its virulence can be exalted by passing through certain animals. Gilbruth has found that by inoculating rats and guinea-pigs with cultures of non virulent or slightly virulent plague bacilli mixed with a streptococcus or the diphtheria bacillus, the animal rapidly died with the post-mortem signs of plague.

Occurrence of the Bacillus in the Body.—The plague bacillus is found in the buboes in large quantities, a smear from the cut surface of the bubo or material obtained from a puncture frequently showing large numbers of the bacilli, which are also found in the œdematous fluid surrounding the inflamed gland. The smear on staining with appropriate methods shows pus corpuscles, cell debris, blood corpuscles, and micro-organisms. During life, except in moribund cases, it is little use examining smears of blood obtained from the peripheral circulation. During the last twenty-four hours of life, when septicæmia has supervened, blood taken from the ear or finger may show plague bacilli.

In pneumonic cases smears from the sputum will generally reveal the bacillus almost in pure culture. It may also be found in the papule or pustule which formed the original point of entry of the bacillus into the system. Post-mortem smears from most of the internal organs, particularly the liver and spleen, show the organism.

Bacteriological Examination of Suspected Material.—After examination of films of the suspected material, if numerous plague-like bacilli are visible, animals (white rats or guinea-pigs) may be directly inoculated. If these bacilli are scanty, it is preferable to make surface cultivations upon agar, and after twenty-four or thirty-six hours, or after making another subculture if necessary, to inoculate a susceptible animal with some of the growth. Intraperitoneal injection of a guinea-pig will produce death or a dying condition in twenty-four hours, with congestion of the peritoneum and viscera, and a characteristic exudation in the peritoneal cavity, grey, turbid, and viscid in character, and containing numerous plague bacilli (Klein, *Public Health*, December 1900).

Subcutaneous injection into the groin of a

guinea-pig or rat produces enlargement of the inguinal glands in twenty-four hours, and usually death in forty-eight to seventy-two hours.

Post-mortem Appearances in Plague-infected Animals.—The appearances in naturally infected rats, mice, and cats are much the same. There is general congestion, œdema, and minute hæmorrhages under the skin and into various viscera, and commonly enlargement of lymphatic glands in different situations, with or without pneumonia.

7. Diagnosis

(1) *Early Recognition of the Disease in a Locality.* — In most towns or places which become plague infected there is well-nigh invariably a section of the press and a large body of the public who most strenuously deny the existence of the disease within their midst, and who place every obstacle within their power in the way of those who are combating the epidemic. This obstruction on the part of the public has been well marked in India, Egypt, and South Africa; in the latter instance not only among the natives and coloured people, in whom ignorance is to be expected, but also among educated Europeans. This attitude is not merely interesting to note, but it is also of importance to recognise, and to expect in future plague epidemics, wherever they may occur, though in this country it is to be expected public obstruction would be slight.

In the early days of an epidemic the only evidence upon which an absolute diagnosis of plague is justified is that of the bacteriologist. Although he can give an opinion on merely staining smears obtained from the patient, he cannot give an absolute diagnosis for three or four days, when his animal experiments are completed. During this period, however, the public health authority has to take precautionary measures, and as far as isolation goes to act as if this suspected case were genuine plague.

The occurrence of two or three cases of pneumonia in the same house or among those visiting it, or several sudden deaths in a neighbourhood under circumstances where association of these persons with one another is known, should cause suspicion as to the presence of some epidemic disease, possibly plague. An increase in the number of deaths in a locality, especially an undue number of certificates of pneumonia, heart disease, and puerperal fever, may become a suspicious circumstance. The occurrence of any illness in a locality in which rats are known to be dying should be looked upon with suspicion until plague can be definitely excluded. Convulsions among children with speedy death may be a manifestation of plague.

(2) *Diagnosis.*—On the occurrence of a suspicious case of glandular fever, after noting the presence or absence of symptoms already described as present in plague, it will be necessary, with antiseptic precautions, to puncture the gland for bacteriological examination. This may be conveniently done by the following procedure:—

(*a*) Wash the skin over the bubo with soap and water, then freely with alcohol. Introduce the needle of a sterilised hypodermic or antitoxin syringe into the periglandular tissue or the substance of the gland and draw off some fluid. It may be necessary to steady the gland, and very little fluid may be obtainable.

(*b*) Make cover-glass smears from the fluid drawn off, and allow them to dry in the air. Fix the films by passing rapidly through the flame of a Bunsen burner or spirit-lamp three or four times.

(*c*) The smears can then be stained, as already described, or transmitted to the bacteriologist by placing them in a small box well padded with cotton-wool.

(*d*) Some of the fluid should also be sent to the bacteriologist, or cultures made directly at the bed-side.

The bacteriologist then makes microscopic, cultural, and inoculation experiments, and a diagnosis is arrived at.

During the course of an epidemic such precautions are unnecessary, and a history of exposure to infection (where obtainable) and a general examination of the patient are usually sufficient. The chief points of importance are —sudden onset, prostration, fever, headache, rapid pulse, the coated tongue with red edges, vomiting, giddiness, thickness and hesitancy in speech, injection of the conjunctivæ, staggering gait, and glandular tenderness. Cases of *pestis ambulans* are difficult to diagnose, and the glands should be punctured where possible. Failure to recover the bacillus does not, however, put plague out of court.

Pneumonic plague can be diagnosed from the prostration and illness out of proportion to the lung changes which are found, the absence of the respiration-pulse ratio in ordinary, but not so markedly absent in plague pneumonia, and the presence of the specific bacillus in the sputum.

Septicæmic plague can only be absolutely diagnosed from an examination of the blood, which usually shows the bacillus in this type of the disease. During the course of an epidemic of plague, especially among races who do not readily consult qualified medical men during their illness, post-mortem examinations should be made wherever possible in cases of sudden death, or if otherwise suspicious.

Scheube states that the serum agglutinative reaction (equivalent to the Widal reaction for typhoid) is constantly absent in the early stages of the disease, where it would be of special

value, and though present in convalescence it cannot be regarded as of any diagnostic help. It has, however, been employed to test suspicious bacilli.

(3) *Differential Diagnosis.*—This may best be considered under the headings of diseases with and without glandular swellings.

(*a*) *Diseases with Glandular Swellings.—Results of strains and injuries* are frequent reasons advanced, especially among native races. In ordinary cases of plague the severe nature of the attack points to some acute fever. Milder cases may present more difficulty, but when in doubt the gland should be punctured and the fluid bacteriologically examined. Although the patient may be able to point to a wound on a limb as a cause of his inflamed gland, it must be remembered that the infection of plague may have entered through this wound. In plague the patient feels very ill before the bubo appears.

Diphtheria and tonsillitis with cervical swellings have been confused with plague, from omitting to carefully examine the condition of the throat. Such examination should always be carried out in cases with swellings in the neck.

Syphilis, gonorrhœa, and sores on the penis may be the cause of the bubo, but examination of the patient clears up the diagnosis. Plague may, however, occur together with these diseases. The hardness of the glands and absence of tenderness in syphilis, and the fever and severe constitutional symptoms in plague, distinguish these diseases. A sore under the prepuce may cause inflamed glands in the groin.

Chronic tubercular glands may be mistaken for plague, but the history, if reliable, is sufficient to distinguish such cases.

Mumps may cause difficulty in diagnosis, but in mumps the bilateral swelling, the metastatic inflammation of the testicle, which, however, occurs late in the disease, the comparatively trivial nature of the disease in children, and the low temperature, and in plague the exquisite tenderness of the gland, the severe nature of the disease, and other symptoms already described, should be sufficient to distinguish them. In plague the parotid gland is not commonly involved, and when it is, the disease is generally very severe, and there is very considerable infiltration of the surrounding tissues. Double parotitis in plague is rare. Cases of mumps do, however, occur with intense prostration and delirium, and mild cases of plague with enlargement in the parotid region. These cases cause difficulty, and recourse would have to be had to the diagnostic puncture.

Filarious lymphangitis has been mistaken for plague, but an examination of the blood for filaria would clear up the diagnosis.

Veldt sores and tic bites may cause inflammation of lymphatic glands. The condition of the patient distinguishes such cases.

Pestis minor has been referred to (p. 118).

Neuralgic pains in the groins are not uncommon among those engaged on plague work. They seem to be usually neurotic, but cause considerable anxiety.

(*b*) *Diseases without Glandular Swellings.—Puerperal fever* may be diagnosed instead of plague septicæmia. In plague, the fever comes on before delivery or abortion, and is almost invariably fatal. The absolute diagnosis of plague rests upon the bacteriological examination of the blood.

Septicæmia and pyæmia can only be differentiated from plague by bacteriological examination.

Smallpox may be confused with plague until the rash comes out.

Influenza, septicæmic and pneumonic plague may present great difficulty in diagnosis; the character of the pneumonia and nervous symptoms are similar in each. Examination of the blood and sputum is necessary.

Cerebro-spinal meningitis and plague both show meningeal symptoms, but in plague retraction of the head is not common. Meningitis may supervene in plague.

Malaria may be confused with plague. The character of the tongue is different; examination of the blood and the treatment clear up the diagnosis.

Typhoid fever may be diagnosed by Widal's reaction, the spots, and other characters of the disease. In doubtful cases bacteriological examination of the blood should be employed. The onset is insidious in typhoid and sudden in plague. In plague the temperature rises suddenly.

Typhus, dengue, and relapsing fever should be differentiated from plague by examination of the blood, together with the signs of these diseases.

Lobar pneumonia may simulate plague pneumonia, but a bacteriological examination of the sputum and the characteristics of these diseases indicate the diagnosis.

8. Prognosis

Plague is a very fatal disease, the case mortality among European patients treated in hospital being about 35 per cent, and much higher among native races.

The prognosis depends on (1) *the period of the outbreak.* Cases are milder at the beginning and end of an epidemic, but not invariably. (2) *On the type of the disease.* Pneumonic and septicæmic cases are generally fatal. (3) *On the age of the patient*; unfavourable in the very young and the aged. (4) *On the site of the bubo.* Cervical lesions are most fatal; this type is frequent in children. (5) *The race of the patient* has an influence. Most of the Asiatic races show a lack of resistance to the

disease. (6) *Sex* has no influence except that if the patient is pregnant she usually aborts or is prematurely confined, with nearly always fatal results to the mother and child. (7) *The stamina of the patient* has an influence, though often absolutely healthy persons contract the disease and die in a few hours. (8) *Haffkine's prophylactic* influences the disease greatly, rendering the type of the disease as a rule mild. (9) *Co-existing diseases*, such as tuberculosis, may make great advances in convalescence, and the patient may succumb to acute phthisis. (10) *Other points* to be noted are—the character of the heart and pulse; obstinate vomiting; early diarrhœa; brain symptoms, such as long-continued sleeplessness, delirium, and coma; hæmorrhages: all these are bad signs. The presence of the bacillus in the blood points to septicæmia and approaching death.

If the patient survive the first week of his attack the prognosis becomes more favourable.

Early hospital treatment gives the patient his best chance of recovery.

9. Treatment.—(*a*) *Symptomatic*; (*b*) *Antitoxic*

(*a*) *Symptomatic treatment* is the chief line to be taken. The *general management* of the case consists in rest in bed, in the recumbent attitude, for at least ten to fourteen days, the patient being on no account permitted to get out of bed. A sufficient supply of nurses should be provided, as a patient during the acute stage should never be out of sight of an attendant. After ten to fourteen days the patient may get up gradually, the condition of the heart and pulse being carefully watched, and provided no complications arise. The patient should be kept in cool and well-ventilated surroundings. Every case should be treated in hospital, not only to control infection as far as possible, but also because of the importance of trained nursing and prompt medical assistance in the sudden emergencies which may at any moment arise.

As regards *the diet*, it should be light and nourishing. Milk, beef-tea, soups, and beaten-up eggs are permissible while the temperature remains high. Then ordinary light diet should be given. In cases of obstinate vomiting, rectal feeding (peptonised milk 2 to 4 oz. in children, given four-hourly) must be employed, the quantity being cautiously increased. If vomiting persists, and even water cannot be retained by the stomach, it is necessary to give "water-feeds" of 3 or 4 ounces, per rectum, about an hour before the milk-feeds. The rectum must be washed out with tepid water every twenty-four hours while this feeding is being carried on. The feeds are best given by a funnel and tubing with a soft catheter inserted in the rectum.

Stimulants, though frequently necessary, should not be exhibited without due cause. The chief symptom calling for stimulants is heart failure and continued high fever. Brandy, if necessary, should be administered by the mouth. In cases where vomiting is absent it is best given as egg-flip, provided the patient can retain and digest it. In cases of collapse, hot brandy and water, hot bottles and blankets, are efficacious. Brandy should not be given per rectum unless absolutely necessary, for it is apt to set up irritation which may render rectal feeding impossible. In cases where vomiting is present champagne should be tried. Strychnine should be given by the mouth alone, or with digitalis as a cardiac stimulant, as indicated by the state of the pulse and heart. But its chief use lies in its hypodermic administration to cases suffering from cardiac depression or sudden heart failure. Here it may be given in doses of 5-10 ♏ every hour, or less frequently, but the patient should be watched carefully to see that no symptoms of poisoning occur. It is surprising, however, the toleration these cases have for strychnine. It is impossible to give definite instructions on this point, as each case must be judged on its own merits, but strychnine is the sheet-anchor of our treatment of plague. In septicæmic and pneumonic plague a few minims of this drug must be administered every few hours, hypodermically if necessary. Ammonium carbonate is an excellent stimulant, especially in pneumonic cases. Ether is very useful in sudden collapse on account of its prompt effect. The nurse must be instructed to give hypodermic injections of ether and strychnine promptly in cases of collapse, as any delay may be fatal.

Restlessness, sleeplessness, and continued high fever should be treated with cold sponging and packs, but in children restlessness is often found to be controlled by hot sponging. Phenacetin and antipyrin should not be used, but $\frac{1}{100}$ grain of hyoscine, or $\frac{1}{8}$ to $\frac{1}{4}$ grain of morphine, may be necessary in some cases.

Vomiting should be treated by hot applications, turpentine stupes, or a blister to the epigastrium. Hydrocyanic acid is frequently efficacious, and often sucking ice gives relief.

The bowels should be kept open by enemata rather than by drugs, but 5 to 10 grains of calomel, if necessary, should be given at the onset of the attack. *Diarrhœa* should be controlled by 10 grains of salol or a starch and opium enema.

The bubo should be treated with hot fomentations, lead and opium, or glycerine and belladonna applications to relieve pain, and should be treated, if it suppurates, on the ordinary lines of dealing with abscesses.

The eye, joint, and other surgical complications should be treated in the usual manner.

(*b*) *Antitoxic Treatment.*—With regard to the serum treatment of the disease, the subcutaneous injection of *Yersin's serum* has not yet been proved to have served much curative purpose, possibly on account of cases not coming under

treatment sufficiently early; possibly, also, the dosage has been sometimes insufficient, but 200 c.c. given subcutaneously have in some cases been found to be of benefit. More encouraging results have been obtained by the intravenous administration of 30 to 60 c.c. of the serum, and this may conveniently be injected into the veins on the dorsal aspect of the hand or the front of the wrist. If the serum be warmed the patient experiences little discomfort.

Lustig's serum has been used in India both subcutaneously (in doses of 200 to 2000 c.c.) and intravenously. It had little effect in septicæmic cases, but appeared to be of some service in bubonic cases with multiple buboes. It cannot, however, be regarded as of much assistance in serious cases. Although the serum treatment of the disease appears to be the only one which holds out much prospect of reducing the case mortality in plague, it must be admitted that as yet the value of the preparations available has not been sufficiently established.

10. Curative and Preventive Sera and Prophylactics

A number of sera and prophylactics have been prepared, and their use advocated by those who have discovered them. The value of some of them is based rather upon laboratory experiments than upon use on a large scale.

(1) *Yersin's serum* has received trial as a prophylactic agent; 10 c.c. are administered subcutaneously into the skin of the abdominal wall, giving an immunity for ten to fourteen days. To obtain further immunity it is necessary to repeat the injection at the end of each period of ten or fourteen days. The use of this serum is only to be recommended as a prophylactic in cases where it is desirable to rapidly immunise a person who has been exposed to infection. In the great majority of cases, as will be seen, Haffkine's prophylactic should be employed. Reference has already been made to the curative use of Yersin's serum. Its mode of preparation is to inject living cultures of plague bacilli intravenously into a horse; the serum is obtained by bleeding the animal.

(2) *Lustig's serum* is obtained from the blood of horses immunised by the injection of a nucleo-proteid, which is prepared by treating plague cultures with 1 per cent caustic potash; after acidifying with dilute HCl the precipitate is filtered off, washed, and dried. The nucleo-proteid is stated to have prophylactic properties. The curative results of the serum have not been very promising in India.

(3) *Terni and Bandi's serum* is obtained by immunising animals with a vaccine prepared by intraperitoneal injection of rabbits with plague bacilli; the peritoneal secretion is treated with "sea-salts" and heated for some hours at 60° C. I am not aware of the results of using this serum and vaccine on a large scale.

(4) *Haffkine's Prophylactic.*—In the preparation of his plague prophylactic Haffkine was led, from the results of his anti-cholera inoculation, "to treat the system with a combination of the actual bodies of the plague bacilli and of the concentrated products of their activity." Plague bacilli are cultivated in broth, from the surface of which they are suspended as a stalactite growth from drops of clarified butter (*ghee*) or of cocoa-nut oil floating on its surface. The flasks containing the growth are periodically shaken, and the growth falls to the bottom, allowing a new crop of stalactites to grow from the surface. An increasing amount of toxine is also formed, secreted from the bacilli and accumulating in the broth. This process is continued for five or six weeks, when it is stopped, and the bacilli killed by exposing the flasks to 70° C. for one hour. The prophylactic is then ready for use after the dose for man has been determined. This has to be done by experiment for each brew of the prophylactic. The fluid is sealed up in vessels, after adding a small quantity of some antiseptic, and sent out for use. The usual dose is about 5 c.c. for an adult, and should be injected into the outer part of the upper arm. A few hours after inoculation, malaise, fever, and aching at the site of injection occur, followed by some prostration, together with redness and swelling of the upper arm. These symptoms usually last about twenty-four hours, when the person inoculated resumes his normal health, though the local inflammation and swelling may persist for some days. Sometimes the reaction is somewhat more severe than has been indicated, and the patient may be some days before he recovers. Rarely abscesses occur, as with any other subcutaneous injection. The most convenient time for being inoculated is in the afternoon, say about 3 P.M.; about 8 P.M. constitutional symptoms occur, the patient passes an uncomfortable and restless night, feels more or less ill next day, which should be spent at rest. On the following day he is well enough to resume his ordinary duties in the majority of cases. A lead and opium application may be of service where there is much local reaction.

Immunity after inoculation, which is never absolute, is established in about ten days (though Haffkine and Bannerman put it at a much shorter period), and persists for a time which is not yet determined; it is at least six months, and probably longer. As the duration of an ordinary plague epidemic is not as a rule longer than this, the person inoculated has secured a large measure of immunity for that particular epidemic, though, if he wishes to continue protected, he must again undergo

inoculation should there be recrudescence of the epidemic. The conclusions of the Indian Plague Commission upon this subject were :—

(1) Inoculation is harmless.

(2) When given in the incubative stage, *i.e.* before the signs of plague are apparent, it has in many cases the power of aborting the disease.

(3) Inoculation affords to all those inoculated a strong protection against attack by plague.

(4) In the few cases where inoculated people are attacked a very large proportion recover.

There is a difference of opinion held by many people as to the second of these conclusions, but it is supported by Bannerman, an authority on the subject. As to the first, third, and fourth of these conclusions, there can be no question of the accuracy of the statement of the Commission.

By again undergoing inoculation ten days after the first injection a greater amount of protection is secured.

(5) *Kitasato's prophylactic* is prepared from agar cultures of plague bacilli. Its inoculation effects are much milder than those of Haffkine's prophylactic. It has been used on a large scale in Formosa with excellent results, both with regard to reduction of the rates of incidence and of case mortality among the inoculated (*Journal Trop. Med.*, May 1, 1902).

(6) *Calmette's vaccine* is obtained from cultures of the bacillus upon gelose. By filtration and repeated washings the bacilli are obtained as free as possible from toxines. The dried bacilli constitute the vaccine, of which the value does not appear to have been tested on a large scale.

11. Measures to be taken upon the Occurrence of an Outbreak of Plague

(1) *Administration*

The public health authorities of a place likely to become plague infected should plan out the details of the arrangements which will become necessary in the event of the arrival of epidemic plague.

A. *Measures in anticipation.*

(*a*) The provision and equipment of a plague hospital and of an ambulance department.

(*b*) The provision of reception houses for "contacts."

(*c*) Arrangements should be made for securing the services of an adequate medical and nursing staff should they be required.

(*d*) The details of the measures to be presently described should be considered, so that they may be ready to be set in motion at the outset of the epidemic.

(*e*) A campaign against rats and insanitary conditions should be at once instituted.

B. *Measures to be adopted at the outset of the epidemic.*

(*a*) The staff of the plague hospital should come on duty and be suitably augmented as required. This includes medical officers, nurses, ward attendants, porters, etc. The menial staff, especially among native races, should be recruited from recovered patients, after the first few weeks of the epidemic. Every plague case should be removed to hospital.

(*b*) The reception houses must be opened.

(*c*) Disinfection gangs must be formed, each in charge of a reliable "ganger," and the whole under some responsible person.

(*d*) The M.O.H. would in this country be in charge of the plague administration, but it may be necessary, especially abroad, to appoint a medical officer in charge of the plague administration.

(*e*) The locality should be divided into districts of a suitable size, each in charge of a medical officer (D.M.O.) with a staff of sanitary inspectors. The D.M.O.s should visit suspicious cases and act as medical referees in conjunction with the medical attendant (if any) in charge of the case. It is convenient, especially in the Colonies, to place the D.M.O.s in sanitary charge of their districts, under the senior medical officer of the plague administration.

(*f*) A bacteriologist is necessary.

(*g*) In some of the Colonies an advisory plague board, composed of men of influence in the locality, has been found of use. This board should not have any executive functions.

(*h*) The plague staff should all be inoculated with Haffkine's prophylactic, and this should be made a condition of employment.

(*i*) All persons employed on plague duty should be segregated and provided with housing accommodation by the plague administration.

(2) *General Measures to be adopted*

(*a*) *Public co-operation* must be secured as far as possible. It usually happens that the inhabitants of a town attacked by plague are divided into two parties—those who are panic-stricken at the advent of the disease, and those who scout the idea of its presence. The former can be soothed, and some of the latter perhaps convinced, by holding public meetings addressed by prominent men in whom they have confidence, also by advertisements, posters, leaflets, and articles in the press. These should advise on preventive measures necessary, and point out the manner in which plague may be contracted and spread. The destruction of rats and a campaign against insanitation should be urged. The importance of early notification, both from the patient's and the public's point of view, should be represented, together with the advisability of seeking early medical assistance in suspicious cases.

(*b*) *Preventive inoculation by Haffkine's prophylactic* must be prominently laid before the public. Its acceptance will largely depend on the confidence with which the people regard

the medical officer who advises it, and also upon the light in which people regard vaccines. We are compelled to admit that this prophylactic is less preventive against plague than vaccination is against smallpox, but the extent and virulence of the epidemic may be sufficient to overcome conscientious objection. Persons exposed to infection by living or working in infected localities should be specially urged to protect themselves by inoculation. The statement of the Indian Plague Commission already quoted should be made public, or some such notice as the following should be widely distributed:—

"Plague Preventive Inoculation

"The public are strongly urged to submit themselves to the preventive inoculation against plague, whereby they will receive a very great amount of protection against the disease. It has been proved of great benefit in India, Australia, and South Africa, where among the small proportion of the inoculated who contracted plague the disease has usually taken a mild course. The operation is followed by some feverish disturbance which passes off in a few hours, and by some swelling of the upper part of the arm, which should be kept at rest for a day or two. Persons may be inoculated by their own medical attendant, or at the following places by the officers provided for the purpose. . . ."

(*c*) *Sanitation.*—A general campaign must be conducted against insanitary conditions, especially against those likely to encourage the presence of rats near houses. Overcrowding should be abated as far as possible, and filthy houses cleansed. Special attention should be devoted to such occupations as rag picking, old clothes selling, and the like. The exportation of rags must be prohibited.

(*d*) *Medical Supervision of Contacts.* — In some places it has been customary to remove contacts from their houses, and to place them in an enclosure under a guard for ten or twelve days. During this period they are daily inspected by a medical officer, and are not allowed to mix with the outer world. This system has been adopted, not only among natives, where it is often inevitable, but also in the cases of Europeans. It imposes great hardships in many instances, and is not a reasonable plague preventive measure where it can be avoided. The idea is to daily inspect every person who has been exposed to plague infection until the expiration of a liberal incubative period. Provided these persons have been compelled to evacuate their houses till these have been disinfected, and provided the clothes they are wearing are also disinfected, I can see no objection to their mixing with the outer world during the period they are under observation. The only necessity is that they should not evade medical inspection, and this could be easily guaranteed upon their own undertaking, in the case of the respectable, and others should be asked to find some surety for their appearance at the daily inspection. In this way a man goes to work from the reception house to which he has been removed, and his mode of life is as little inconvenienced as possible. There is a difficulty in carrying out such a scheme among native races, but every effort should be made to avoid compulsory segregation of contacts, which has been one of the great sources of public opposition to plague measures.

(*e*) *Evacuation of Infected Premises.* — All premises, *whether infected by rat or man*, should be forthwith evacuated and disinfected. A camp of tents or other dwellings should be provided, into which the inhabitants of blocks of buildings should be sent while their houses are being disinfected. Contacts of cases should be sent to reception houses or camps of this nature and daily inspected. It is essential to remove every plague case, except those who are practically moribund, to hospital.

(*f*) *Disinfection; Disinfection of Infected Premises.*—Infected premises, being defined as those infected by man or rats, must after evacuation be disinfected. This cannot be done whilst the people are in their houses. They can be despatched to the reception house to undergo personal disinfection, and remain there or with friends till their houses are ready for them. After removal of the case and contacts, the house should be taken in charge by the plague authorities and locked till the disinfecting gang arrive. It should then be subjected to fumigation by burning sulphur, liquefied SO_2, or by some efficient type of formalin lamp. After twelve hours the house should be opened, and after an inventory is taken, the interior of the dwelling cleansed by washing and spraying the walls and ceilings with 2 per cent formalin solution or corrosive sublimate. A useful stock solution is—Corrosive sublimate, 4 oz.; hydrochloric acid, 1 pint; water to 1 Winchester quart; colour with litmus. For use, add half a bottle to 6 gallons of water, or 8 fluid oz. to 1 gallon of water. This must be mixed in wooden vessels. The furniture should be sprayed with the 2 per cent formalin solution.

A certain number of articles, such as filthy rags and blankets, must be burnt, and such things as bedding, clothes, etc., sent to the steam disinfector. After the house is disinfected it must be exposed to the influence of sunlight and fresh air. The point in which the disinfection of a plague house differs from that of any other infective disease is that it is absolutely necessary to remove all floor, skirtings, false ceilings, and so on, if there are any signs of rats about the house. This causes considerable expense and takes time, but it has to be done thoroughly. Any dead rats found must be at once cremated, and the infected area saturated

with disinfectants, such as Jeye's fluid (2 oz. to 1 gallon of water). Rat runs must be followed, dug up, and obliterated. Broken glass, tar, and crude carbolic acid (Calvert's No. 4) are useful in this connection. Basements of houses, and the floors of stores of grain and forage, should be concreted and made as rat-proof as possible.

All out-buildings, cellars, etc., should be lime-washed (unslaked lime, ½ lb. to 3 gallons of water), and the opportunity should be seized of requesting the landlord, or by serving him with the proper notices, to make any necessary alterations to the house to put it in a satisfactory sanitary condition.

As regards disinfection of native huts in South Africa, India, and elsewhere, mud floors should be disinfected with phenyle (1 to 50) or acid corrosive sublimate ($HgCl_2$ 1 part, strong HCl 2 parts, water to 500 parts). In many cases it is possible to burn down the huts. Impermeable floors should be disinfected with phenyle (1 to 100) or acid perchloride of mercury (1 to 1000).

Disinfection of clothes must be effected by steam where possible, or by destroying them by fire if worthless. If this cannot be done they should be fumigated, boiled, immersed in disinfectants, exposed to sunlight, or disinfected by the best method available.

Disinfection of ships is best effected by sulphur fumigation, as described under "destruction of rats on board ship" (p. 129). Infected quarters should be fumigated, and the woodwork washed down with acid corrosive sublimate solution, the bedding, etc., being removed, if possible, to a steam disinfector.

Disposal of the Dead.—If death occurs at home, the body must be at once handed over to the plague authorities, wrapped by them in a sheet soaked in corrosive sublimate, placed in a coffin, screwed down, and conveyed to the place of burial, which must be adjacent to the plague hospital. Wakes must be strictly prohibited, and the aid of the Roman Catholic clergy should be sought for this purpose. Cremation, when possible, should be employed.

Disposal of Excreta and Discharges.—These should be received into 5 per cent carbolic acid solution and disposed of by burning in an incinerator, or by heating them to 100° C. in a large iron pot. After complete chemical disinfection they may be disposed of by burial or by the sewerage system.

(*g*) *Inspection of Travellers by Rail.*—This has been conducted in many places abroad, and no one has been permitted to travel until they had received a certificate that they presented no signs of plague. It has prevented many infected natives in India from travelling. In this country some system of notification of departure from an infected locality might perhaps be usefully employed, and sent to the local authority at the passenger's destination.

(3) *Destruction of Rats.*—(*a*) On land; (*b*) On board ship.

(*a*) *On Land.*—The various methods of clearing premises of rats, such as employing professional rat-catchers with ferrets and dogs, or laying down poison and traps, must be adopted. It must be remembered that ferrets are liable to contract plague. Keeping a mongoose on the premises has been highly recommended, especially in places where the mongoose cannot readily escape. Poisoned rats cause a certain amount of nuisance, but the smell can be traced, and by taking up the floors and so forth their haunts can be discovered and the rat runs obliterated. When premises have been cleared of rats the necessary steps must be taken to prevent them gaining entrance again, by stopping up or wiring any apertures which might serve as a run, and by concreting where necessary, especially in stores and basements. Doors must be made to fit accurately, and to be bound in iron at the bottom if possible, to prevent rats gnawing their road in. The process therefore consists of two actions: (1) a simultaneous campaign against rats all over a town; (2) making premises rat-proof, after they have been cleared of rodents. Unless the public take the matter up little can be done.

Danysz's Method.—J. Danysz, chef de laboratoire à l'Institut Pasteur, has exalted the virulence of an organism of the *coli* group, which, under certain conditions, causes among rats and mice an epidemic disease which is not transmissible to man. This organism, morphologically identical with the *Bacillus typhi murium* (Löffler) and with Gaetner's bacillus, can be made pathogenic for all varieties of rats with some difficulty. Danysz has described the method by which he has done this in the *Annales de l'Institut Pasteur* for April 1900, one feature being the growth of the culture in collodion capsules in the peritoneal cavity of the species of rat whose extermination is being sought. The practical application of Danysz's virus is to spread growths of the bacillus on bread and to place it where rats frequent. An epidemic is claimed to be set up, which, after three or four passages through these rodents, finally dies out. The method has its limitations, and has been of little value in some places where it has been employed. Danysz recommends that it should be used in spring and autumn, so that the young rats, which are more susceptible, should be destroyed. It is also important to keep on laying down fresh cultures to replace those whose virulence has died out. Danysz is of opinion that by systematic attack for a season or two on the young susceptible rats the complete extirpation of these animals may be secured. Klein and Williams, working on this bacillus both in the laboratory and on a large scale, arrived at negative conclusions as

to its value (*Lancet*, Aug. 17, 1901). The method is far from being perfected or certain in operation, and much further laboratory research is necessary, coupled with experiments on a large scale. It would seem to be necessary to exalt the virulence of the organism locally, by experiment, so that it should become capable of destroying the race of rats which infest the neighbourhood in which it has to be employed.

(*b*) *On board ship* rats can only be destroyed to any great extent when the vessel is *empty*. Then by sulphur fumigation of the compartments a great proportion of the rats can be destroyed. In Australia there is an intercolonial agreement under which vessels are compelled to produce a certificate of fumigation given by officials provided for the purpose, and without which they are liable to detention. This method is stated not to unduly interfere with trade, and to be in practice not difficult to secure. This answers some of the great objections to the compulsory destruction of ship rats. It is obvious that the only way to prevent plague rats gaining access to an uninfected port is to destroy, systematically, rats, healthy or otherwise, on board every ship, as far as possible. It is further necessary, if the ship is to be kept rat free, or as nearly so as possible, to keep the vessel moored some distance off the dock, to hoist in gangways at night, and to provide guards on the ropes and hawsers, and by tarring them, to prevent communication of ship and shore rats. It is of importance to recognise that rats enter and leave ships in cargo such as forage and skeleton crates, even when the vessel is lying out some way from the shore, and cargo is brought off in lighters, so that fumigation of the vessel when empty is necessary at the end of each voyage.

The generation of CO_2 in the holds of ships has been advocated, but from the lack of penetrating power of this heavy gas the method has not attained much success. The method of sulphur disinfection which appears to work best in practice is that produced by the *Clayton machine* (of the Clayton Fire Extinguishing and Ventilation Company), which consists of a special apparatus. It drives the gas produced by the combustion of sulphur into the lower parts of the holds of ships, made as nearly air-tight as possible, while air is extracted from the upper part till the whole of the air space contains about 10 per cent SO_2. The air extracted is passed over the heated sulphur in the furnace. The apparatus may be placed on deck or on a barge alongside, so there is no danger of causing a fire in the hold. The quantity of sulphur used is 1 lb. per 400 cubic feet of space, and the cost is about 20s. per 100 tons gross register (*Public Health*, March 1902). This "Clayton gas" appears to owe a considerable amount of its efficiency to the presence of some SO_3 which is formed, in addition to the SO_2, by the high temperature in the furnace. The results obtained are vastly superior to those involving the use of pure SO_2. It is stated that less than 5 per cent Clayton gas is as sufficient as 15 per cent pure SO_2 in its power of destroying rats and other vermin. The Clayton gas has, it is said, no appreciable action upon coloured materials, food-stuffs, paints, or dry metals, except in a moist atmosphere. Further, 8 per cent of this gas is stated to rapidly extinguish burning materials. At Cardiff recently it was found that by this method rats placed in cages at the bottom of the hold were killed almost immediately, and those about 6 feet below the level of the deck within three hours' exposure. As far as disinfection went, liquid cultures of plague, cholera, and typhoid, soaked in strips of fabric were killed after an exposure of twelve hours, but anthrax and one of three cultures of *staphylococcus pyogenes aureus* remained alive (*Lancet*, June 21 and July 19, 1902).

(4) *Measures Employed in excluding Plague from a Country.*

(*a*) *International Notification.*—This is done among the signatories of the Venice Convention of 1897, which includes the leading European powers, but does not include Portugal, Servia, Turkey, and Greece. Notification takes place upon the occurrence of epidemic plague in man, but no steps are taken upon the outbreak of the disease in rats, an omission of considerable importance in the light of our present knowledge.

(*b*) *Arrest of Infected Persons and Things.*—Under the cholera, yellow fever, and plague order of the L.G.B. (England) 1896, and of the L.G.B. (Scotland) 1898, all vessels from infected ports are boarded by the port M.O.H. on arrival in this country, and the crews and passengers are medically inspected. If all are well on board, or no suspicious case or death has occurred on the voyage, the vessel is then at liberty to land cargo and passengers. If it is infected, the vessel is required to be moored at a certain fixed place, and the cases or "suspects" removed to hospital. It is to be noted that a "suspect" cannot be detained longer than two days, unless a definite diagnosis of plague is arrived at. In some cases this is too short a time to arrive at a definite diagnosis. The other persons on the vessel are allowed to land after furnishing their names and addresses, this information being forwarded to the respective sanitary authorities at the destinations. Clothing likely to have been infected, and the infected parts of the ship, are disinfected under the orders of the M.O.H. Where a ship is not ascertained or certified to be infected, but has passengers on board who are in a filthy or otherwise unwholesome condition, *or* has come from an infected place, the M.O.H. may, if in his opinion it is desirable, give a certificate that

these persons be not allowed to land till they have satisfied him as to their names, places of destination, and addresses at such places. The M.O.H. has therefore power to detain such persons for inquiries, and part of such time must be utilised in cleansing their persons and possessions.

(c) *Destruction of Rats afloat.*—The importance of this measure, and the way in which it may be done, has been already referred to. The Local Government Board of England have issued a special memorandum on ship-borne plague and rats, dated April 1901, advising local authorities to destroy rats in infected and suspected ships, to keep them moored off the shore, and to take the same precautions in case of rat mortality in vessels from infected ports. They also *advise* disinfection of ships with infected rats, and that the destruction of all rats on ships and shore should be attempted. This memorandum is of the nature of advice rather than a regulation.

In Bristol and other large ports the following methods are in use:—

(1) A list of infected ports is prepared by the Medical Officer of Health and kept up to date.

(2) An infected port is kept on the list for three months after official clearance.

(3) Vessels, especially grain and cotton-seed boats, from infected or suspected ports, are upon arrival treated as "suspects," moored six feet off the shore, with cables guarded and gangways up at night. Day and night watchmen are employed to prevent rats gaining the shore.

Dr. Davies, in an article "Exclusion and Control of Plague" (*Public Health*, March 1902), puts the matter in a nutshell when he states: "The present policy of allowing the approach of rat-infected ships, and then attempting to prevent the rats coming ashore, is surely attacking the evil at the wrong end. If possible, the obviously correct method would be to insist upon the destruction of all rats in ships when *empty*, and upon the subsequent observance of precautions to prevent the entry of more rats when loading at infected or other ports."

Planarthragra.—Wandering gout, or gout which first attacks one and then another of the joints (Gr. πλανάω, I wander, ἄρθρον, a joint, and ἄγρα, a catching or seizure). *See* Gout.

Plane.—A flat smooth surface (*e.g.* the inclined planes of the pelvis), or, more often, an imaginary plane surface (*i.e.* one in which every straight line joining any two points in it lies wholly in it) used as a standard of reference for the positions of the bodily organs (*e.g.* the plane of the pelvic brim, etc.).

Planicipital.—Flat-headed (Lat. *planus*, flat, *caput*, head).

Plano-.—In compound words *plano-* (Lat. *planus*, flat; Gr. πλανος, wandering) means either flat or wandering; *e.g.* a *plano-concave* lens is one flat on one side and concave on the other, and *planocyte* means a wandering cell.

Planodia.—A false or wandering passage, such as may be made by a catheter during forcible attempts to introduce it into the bladder.

Planomania.—A monomania for wandering, usually combined with a tendency to become unsocial in habits.

Plantar.—Relating to the sole of the foot (Lat. *planta*, the sole), *e.g.* the plantar arch, artery, reflex, or fascia. *See* Ankle-Joint, Region of, Diseases (*Gonorrhœal Affections, Plantar Neuralgia*); Aneurysm (*Lower Limb, Plantar Arteries*); Deformities (*Flat Foot*); Fascia (*Contraction of Plantar Fascia*); Osteo-Arthropathies (*Tabetic Foot*).

Plantaris.—A muscle arising near the knee-joint, and inserted into the os calcis.

Planuria.—The discharge of urine through a fistula or other anomalous passage.

Plaques.—Patches, *e.g.* the *opaline plaques* or white spots due to the action of nitrate of silver on mucous surfaces, or the *plaques jaunes* (yellow patches) seen on the cerebral convolutions in cases of thrombosis in elderly persons. *See* Syphilis (*Secondary Mucous Membranes*); Brain, Affections of Blood-Vessels (*Thrombosis*).

Plasma.—The blood or lymph without the corpuscles belonging thereto. *See* Blood (*Plasma and Serum*); Physiology, Blood and Lymph (*Plasma*). The plasma is the blood *minus* the corpuscles, and the serum is the plasma *minus* fibrinogen. *Muscle plasma* is the juice expressed from muscle. The germ plasma or germ plasm is that part of the substance of the germ cell which is effective in perpetuating the species; a part of it is supposed to be passed on from one generation to another without entering into the formation of the organs and tissues of any one generation (continuity of the germ plasm).

Plasmin. *See* Immunity (*Bacterial Plasmin*).

Plasmodium.—A mass of protoplasm (or syncytium) in which the cell outlines have disappeared (or have not been differentiated), leaving, however, the nuclei distinct; also the name given to the protozoan parasite of malaria. *See* Physiology, Protoplasm (*Structure*).

Plasmogen.—The germ plasm, formative protoplasm, or bioplasm.

Plasmon.—A proprietary preparation made from the proteid of milk. *See* INVALID FEEDING (*Milk*).

Plasmotropic Action.—Blood destruction or degeneration due to the action of poisons, *e.g.* the blood derivatives absorbed from the intestine in ankylostomiasis.

Plasson.—The protoplasm of the non-nucleated cell in which no cell wall has developed; undifferentiated protoplasm.

Plaster.—In Pharmacy a plaster (or plaister) consists of a solid or semi-solid adhesive substance spread upon a piece of muslin or skin; it is used as a means of applying medicines to the skin, of supporting underlying parts, or of closing wounds. *See* JACKET, PLASTER; PLASTER OF PARIS; ETC.

Plaster of Paris.—A white plaster consisting of gypsum (sulphate of lime), which, when mixed with water, swells and quickly sets; it is used for taking casts and for forming a casing to keep injured parts (fractured or dislocated limbs) immobile; it is so named because the gypsum used to be obtained near Montmartre, Paris. *See* BANDAGES (*Fixed*); SPINE, SURGICAL AFFECTIONS OF (*Spinal Caries, Treatment*).

Plastic.—In Surgery *plastic* means reparative, *e.g.* a *plastic operation* (*i.e.* one for the repair of injured or destroyed parts); in Pathology it means formative or capable of being organised, *e.g. plastic lymph, plastic bronchitis*; in both meanings the root is the same, viz. Gr. πλασσειν, to mould or form. *See* BRONCHI, BRONCHITIS (*Clinical Varieties, Plastic*); NERVES, PERIPHERAL (*Injuries, Treatment*); PALATE (*Cleft, Treatment*); SKIN GRAFTING AND ALLIED PROCEDURES.

Plasticine.—A proprietary preparation which can be easily moulded or modelled; it can be used for teaching purposes in Anatomy and Surgery, etc.

Plastid and Plastidule.—A small mass of protoplasm; the unit or ultimate particle of living matter (hypothetical).

Plastin.—The chief proteid of protoplasm; it contains phosphorus.

Plastoprolepsis.—Premature development of a part.

Plastron.—The sternum with the attached costal cartilages as it is separated and raised from the chest in making a post-mortem examination.

Plate.—A flattened structure, such as a thin layer of bone (*e.g.* the orbital plate of the frontal bone) or of forming substance (*e.g.* the mesoblastic plates of Embryology).

Platelet.—A small plate, *e.g.* the circular or oval bodies found in the blood, which are about one-third the diameter of the red corpuscles. *See* PHYSIOLOGY, BLOOD AND LYMPH (*Cells of Blood, Platelets*).

Plathelminths.—Flat-worms. *See* PARASITES (*Helminths, Plathelminths*).

Platiculture.—The development of bacteria on plates.

Platodes. *See* PARASITES (*Helminths, Plathelminths*).

Platy-.—In compound words *platy-* (Gr. πλατύς, broad) has the meaning of flat or broad, *e.g. platystomous* (having a broad mouth) and *platyurous* (having a flat tail).

Platycephalic.—Having a broad or flat head; having a cranium with a vertical index of less than 70.

Platycnemic.—Having a broad or flattened tibia (Gr. κνήμη, the tibia).

Platyhieric.—Having a broad sacrum (Gr. ἱερόν, the sacrum) or one with an index exceeding 100. (The index is got by multiplying the breadth by 100 and dividing by the height.)

Platymesaticephalic.—Having a head which is both platycephalic and mesaticephalic.

Platypellic.—Having a flat pelvis, *e.g.* one with a brim-index under 85° (Gr. πέλλα, a wooden bowl).

Platypygous.—Having broad buttocks (Gr. πυγή, the buttocks or rump).

Platyrrhine.—Having the nose broad and flat, the nasal index being from 51 to 58; also the name applied to the apes of the New World, which have the nostrils wide apart and the thumbs non-opposable or nearly so.

Platysma.—A thin, broad layer of muscle found immediately below the skin of the neck in the human subject; it represents the *panniculus carnosus* of animals.

Playfair Treatment. *See* NEURASTHENIA (*Treatment, Weir-Mitchell Method*).

Plenum System.—A propulsion system of ventilation. *See* VENTILATION AND WARMING (*Plenum System*).

Pleo- or Pleio-.—In compound words *pleo-* or *pleio-* (Gr. πλεῖον, more) has the meaning of "more than one," *e.g. pleiomastia* and *pleio-*

mazia (having more than the normal number of breasts or of nipples).

Pleomorphic.—Having more than one form, *e.g.* certain bacteria or fungi which have different forms at different ages.

Pleonexia.—Selfishness, greed, or avarice as a symptom of insanity (Gr. πλεονέκτης, one who claims more than his share).

Pleonotus.—The type of malformation in which an accessory auricle is found on the neck; cervical auricle.

Plethora.—A morbid state in which the body or a part of it is believed to be abnormally full of blood or of some other humour (Gr. πληθώρη, fulness); in modern usage it means an excess of red corpuscles in the blood, or polycythæmia.

Plethysmograph. — An instrument for measuring changes in the volume of a part of the body caused by variations in the amount of blood contained therein, etc.; it was invented by Mosso of Turin about 1870. *See* Physiology, Circulation (*Mean Blood Pressure, Plethysmograph*).

Pleura, Diseases of.

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Anatomy.—The pleuræ are two independent serous sacs which do not communicate with each other. Each sac consists of two layers—a visceral layer and a parietal layer—the former of which completely invests the corresponding lung, including the sulci between the lobes, while the latter lines the ribs and intercostal spaces, invests the upper surface of the diaphragm, takes part in the formation of the mediastinum, and is continued through the upper aperture of the thorax into the neck. The two layers of the pleura move freely upon each other in health. In disease these movements are impeded or rendered painful. The two layers of the pleura are continuous with each other at the root of each lung, and also by means of the *ligamentum latum pulmonis*, a short double layer of serous membrane which occupies the interval between the root of the lung and the diaphragm. The shape of the pleuræ is not the same on the two sides, being determined by that of the corresponding lung. Inferiorly the lungs do not completely fill the thoracic cavity, except in exceptional cases of extreme inspiratory effort.

Only the relations of the pleuræ which are of clinical importance can be here considered. The height to which the pleuræ rise in the neck shows considerable variations. From half an inch to an inch and a half above the clavicle may be taken to represent the usual limits of this variation. In front the pleuræ of the opposite sides are in contact behind the body of the sternum, but the line of reflection is not constant. It is usually behind either the body of the sternum or its left edge. Inferiorly the relations of the pleuræ to the diaphragm are of great clinical importance. According to Quain, "the lower border of the costal pleura is re-

flected on to the diaphragm, opposite a line passing from the lower end of the sternum outwards behind the seventh costal cartilage, nearly as far as its rib; here it leaves the seventh cartilage, and, continuing to pass obliquely downwards and backwards, crosses the eighth, ninth, tenth, and eleventh ribs, and reaches the twelfth rib near its vertebral end." Others place the inferior limit of the pleuræ in front, at the level of the sixth intercostal cartilage or the sixth intercostal space. In the mid-axillary line the lower limit of the pleuræ is from two to three inches above the lower margin of the thorax. The inferior limit is usually lower on the left side than on the right, viz. the lower border of the ninth rib on the right side, and the tenth on the left. The inferior limit of the pleuræ posteriorly varies from the upper border of the twelfth rib to the level of the first lumbar vertebra. The relations of the pleuræ to the pericardium are sometimes of clinical importance. "The layers forming the sides of the anterior mediastinum pass backwards from the sternum to the pericardium in close relationship with one another, except below, where a triangular interval is sometimes formed between them. At the front of the pericardium the two layers separate, each passing round its own side of the pericardium to the front of the root of the corresponding lung, where it becomes continuous with the visceral pleura" (Quain). The triangular interval here mentioned corresponds to the inner end of the fifth left intercostal space, and is of importance inasmuch as this is the only situation in which *paracentesis pericardii* can be performed without wounding the pleura. In many cases, however, this space is covered by pleura.

Communications by means of the lymphatic channels exist between the pleuræ and the peritoneum.

Pathology.—The pathology of affections of the pleura is at present in an unsettled state. The tendency of recent investigations has been to regard all pleurisies as toxic, to accentuate the large part in causation which is played by various micro-organisms, to regard most pleurisies not otherwise obviously accounted for as probably tubercular, and to call in question the existence of the so-called "simple," "idiopathic," or "inflammatory" pleurisy. These controverted questions will be presently considered in some detail. The bacteriology of the disease has engaged the attention of a large number of observers in this country, on the Continent, and in America, amongst whom may be mentioned Fraenkel, Netter, Weichselbaum, Laveran, Serafini, Levy, Lemoine, Aschoff, Mitchell Prudden, Sidney Martin, Washbourne, Goldscheider, Jakowski, Thue, and Landouzy. The results obtained have been of much interest and considerable practical value, but discrepancies appear in the conclusions of different observers, and it is evident that finality is as yet far from having been attained. The conclusions of pathology, and especially of bacteriology, must be subjected to a rigid criticism and interpreted in the light of the facts of clinical experience before satisfactory views can be attained. In view of the contradictory results of many observers, the statement of E. Levy that "die Aetiologie der Pleuritis welche vor wenigen Jahren noch in tiefes Dunkel gehüllt war, heute mit Hilfe der bacteriologischen Forschung ein wohlgekanntes wohl durchgearbeitetes Gebiet geworden sei," must be pronounced premature. Bacteriology has not yet spoken its decisive word on this subject, but the path of probable fruitful progress has been clearly indicated.

The changes which take place in pleurisy are in many respects those common to inflammations of serous membranes. The membrane becomes injected and hyperæmic, and loses its normal glistening appearance. There is proliferation of the nuclei of the endothelial cells, and soon a fibrinous exudation appears on the surface of the membrane and forms shaggy projections from it. The process may stop at this point, as in the so-called "dry" pleurisy, and adhesions may form between the opposed pleural surfaces; but in a large proportion of cases serous fluid is exuded, and collects between the layers of the pleura. This fluid may consist of limpid serum, or of a more or less turbid and flocculent fluid, or of pus, or it may contain varying quantities of blood. The quantity may vary from a few ounces to many pints. After a variable period absorption usually sets in, and may proceed satisfactorily until convalescence is completed. The two layers of the pleura again come into contact, granulation tissue is formed, and the two layers become in many cases more or less adherent, the pleural cavity being to this extent obliterated. The lung on the affected side tends to collapse, at first owing to the alteration of the normal negative pressure in the pleural cavity, later in consequence of the pressure of the increasing exudation. It may become entirely compressed and airless, or it may float on the surface of the fluid. It tends to shrink towards its root.

We shall now consider the various kinds of effusion in more detail.

A. *Serous Effusion.*—The term "serous" is a misnomer, as the fluid always contains fibrin. It is in reality a dilute liquor sanguinis. The colour is yellow or yellowish green. The specific gravity usually exceeds 1018, and may be as high as 1030, but may also be low. The reaction is alkaline. The fluid may be perfectly limpid, but is often turbid, owing to the presence of large quantities of corpuscular elements. In this latter case the transition to pus is probable, but perfectly limpid serum rarely becomes purulent. Microscopic examination shows the

presence of scattered red blood corpuscles, leucocytes, fatty globules, and endothelial cells. Micro-organisms are often present. Sugar is always to be found in small quantity, and sometimes acetone and uric acid. Serous effusions are highly albuminous, proteid material being abundantly present, chiefly in the form of serum albumin and globulin. Serous effusions coagulate on standing for a few hours. The differentiation of "serous" pleural effusion from passive transudations, due to cardiac or renal disease, may be important. The distinction is not always easy, and may be very difficult or even impossible. The broad rules are that transudations are usually of lower specific gravity than effusions, they exhibit fewer tissue elements, and coagulate with greater difficulty. (See "Examination of Pathological Fluids," vol. iii. p. 300 *et seq.*)

B. *Purulent Effusion* (*Empyema*).—This type of effusion is commoner in the child than in the adult. It is often a sequel of pneumonia, and seems in a considerable proportion of cases the result of invasion by the pneumococcus. It does not often supervene upon a serous effusion unless that effusion has from the outset contained a large proportion of corpuscular elements. It may follow upon repeated tappings of a recurring serous effusion, but this is much less frequent than has been supposed. The pus only exceptionally becomes fœtid.

C. *Hæmorrhagic Effusion.* — The blood is mixed in varying proportion with serum, less often with pus. Hæmorrhagic pleural effusions are somewhat rare, but their importance is great. They are most often due to tuberculosis, less often to scurvy, hæmophilia, one of the malignant exanthemata, Bright's disease, cirrhosis of the liver, or to cancer. It is important to bear in mind that the effusion in tuberculosis of the pleura is most often serous, only exceptionally purulent or hæmorrhagic. In cancer the effusion is often hæmorrhagic, but may be serous. Apart from tuberculosis, cancer, scurvy, or hæmophilia, a hæmorrhagic effusion points to a state of profound dyscrasia or to a virulent attack of one of the exanthemata. A hæmorrhagic effusion in tuberculosis does not necessarily exclude a fairly hopeful outlook. While hæmorrhagic effusions are distinctly unfavourable, their prognostic indications vary widely and need be interpreted with caution. Cancer cells are sometimes present in a hæmorrhagic effusion. According to von Jaksch, the presence of the glycogen reaction in a marked degree favours the suspicion of cancer.

D. A chylous effusion, due to rupture of the thoracic duct, has been described. It is extremely rare.

Bacteriology of Pleural Effusions.—This subject is at present exciting great attention, and may be expected to throw much light upon the pathogeny of pleurisy. The results hitherto obtained are interesting and valuable, but inconclusive. We shall consider the subject under the following heads:—

1. In what proportion of cases of pleural effusion are bacteria present?

2. What kinds of bacteria are found, and what is their significance?

3. What conclusion is to be drawn in cases where the effusion is "sterile"?

As regards the first question we find divergent results. Lemoine found 28 out of 32 cases of serous effusions "sterile." Mitchell Prudden found micro-organisms in only 2 out of 21 cases of pleural effusion. Aschoff found micro-organisms in only 7 out of 200 cases of serous effusion. On the other hand, Thue found micro-organisms present in 12 cases out of 30 in serous effusion, while Jakowski examined 30 cases of serous effusion and discovered micro-organisms in all but 7. It is needless to observe that in cases of empyema micro-organisms are practically always found present.

As regards the second question no constant results have been obtained. Lemoine found the *staphylococcus albus* in 4 cases. He failed to find any micro-organisms in any of his cases which were certainly tubercular. Mitchell Prudden found the *pneumococcus* present in 2 cases of serous pleural effusion following pneumonia. He examined 24 cases of empyema, and found that in "simple" empyema the organism most often present was the *streptococcus pyogenes* (7 times out of 8), while in pneumonic empyema he found the *pneumococcus* in 9 cases out of 11. In one case of tubercular empyema he found the *tubercle bacillus* alone present. Goldscheider found the *streptococcus* in 3 cases of serous pleurisy which did not become purulent, and the *staphylococcus* in 1 case. Aschoff found the *streptococcus* once and the *pneumococcus* once each in 2 cases of serous effusion following pneumonia. In 2 cases of so-called "idiopathic" pleurisy he found the *streptococcus* once and the *pneumococcus* once. In a case of exudation associated with pulmonary gangrene he found the *streptococcus*. Jakowski examined 48 cases of exudation in which micro-organisms were present. In 34 of these only one micro-organism was present; in 10 of these it was the *streptococcus pyogenes*, in 2 the *bacillus tuberculosis*, in 1 the *staphylococcus aureus*; in the remaining 14 cases various micro-organisms were associated. Thue found the *bacillus tuberculosis* present in 1 case out of 12 cases of serous effusion; in the remaining 11 various cocci were found. In 23 cases of empyema he found the *pneumococcus* in 14. Netter examined 110 cases of purulent effusion. He found the *streptococcus pyogenes* in 51, the *pneumococcus* in 32, the *bacillus tuberculosis* in 12, and various bacteria of putrefaction in 15.

Fraenkel, Weichselbaum, Netter, Serafini, and others assign a very important rôle to the

pneumococcus in the ætiology of acute pleurisy. Netter, Laveran, and others regard *streptococcus* pleurisy as much more unfavourable than *pneumococcus* pleurisy. The rarity of the *bacillus tuberculosis*, even in cases undoubtedly tubercular, is noteworthy. Gilbert and Lion failed to find it in any of their 20 cases, though some of them were clearly tubercular. According to von Jaksch, "tubercle bacilli are only present when a discharge of tubercular matter has taken place into the pleural cavity."

As regards the interpretation of those cases of serous effusion which are found to be sterile, there is a growing tendency with bacteriologists to regard them as tubercular. This view rests chiefly on the results of inoculation experiments. Aschoff injected pleuritic fluid from 57 cases of serous pleurisy into the abdominal cavity of guinea-pigs. In 19 of these cases, in which the exudation occurred in connection with pneumonia, rheumatism, and a number of other non-tubercular affections, none of the guinea-pigs became tubercular. In 12 cases of certain tubercular pleurisy the animals becamc tubercular in 7. In 12 cases, doubtfully tubercular, the animals became tubercular in 9. In 12 cases of so-called "idiopathic" pleurisy tuberculosis followed the injections in 9. The author concludes that the majority of cases of so-called "idiopathic" pleurisy are tubercular. Netter puts the proportion of cases of serous pleurisy due to tuberculosis at 68 per cent, Landouzy at 98 per cent. Germain Sée believes that "so-called simplc pleurisy from a chill is only a tuberculous pleurisy, the nature of which has been misunderstood." The whole subject of the relation of pleurisy to tuberculosis—one of the most important problems in practical medicine which are still *sub judice*—can be considercd more conveniently after we have dealt with the clinical aspects of the disease. It must suffice here to indicate the general nature of the bacteriological evidence and the consensus of opinion amongst the pathologists that the connection between the two diseases is an intimate one. Jakowski sums up his conclusions on the entire subject of the bacteriology of pleurisy as follows: —(1) That all pleurisies depend upon micro-organisms, though these are not always to be found; (2) that where no organisms are found the case is probably tubercular; (3) that primary genuine pleurisy, not due to tuberculosis, is most often due to the *pneumococcus*; (4) that serous exudations in which pyogenetic cocci are found have a great tendency towards empyema; (5) that exudations during or after pneumonia are most often dependent on Fraenkel's diplococcus. It is evident that these views are still to some degree hypothetical. See "Bacteriology of Pneumonia," p. 157.

State of the Blood in Pleurisy.—The blood in uncomplicated cases of pleurisy does not show any very marked changes. Houston, working in Lorrain-Smith's laboratory, found a moderate leucocytosis during the pyrexial stage, the leucocytes numbering from 8000 to 12,000 per cm. This leucocytosis passes off as the fever abates. The same observer found during the afebrile stage the following averages in eight cases of uncomplicated pleurisy:—

Red cells . . .	4,100,000 per cm.
White cells . .	8,000 „
Hæmoglobin . .	78 per cent.
Differential count:—	
Polymorpho-nuclear elements . .	= 67 per cent.
Small lymphocytes .	= 28 „
Large lymphocytes .	= 2·8 „
Transitional leucocytes . . .	= 1·8 „
Eosinophile leucocytes . . .	= ·4 „

In the febrile stage the proportion of polymorpho-nuclear elements in the differential count is about 70 per cent or more.

Houston is of opinion that the blood-count may be of value in excluding other conditions. In empyema, for example, the leucocytes are usually between 18,000 and 30,000 per cm. In pneumonia a similar condition is found, while in malignant disease of the lung a large leucocytosis has generally been recorded.

In cases of pleurisy the red corpuscles and amount of hæmoglobin usually show only a slight decrease. In cases of chronic pleurisy and empyema the red cells may fall as low as 2,000,000 per cm., and the hæmoglobin may fall to 35 per cent. An empyema may disappear after tapping, and the absence of a leucocytosis has been found useful in determining whether the suppurative process has ceased or not.

Acute Pleurisy

Ætiology.—The part played in the causation of pleurisy by various micro-organisms—especially the *bacillus tuberculosis*, the *pneumococcus*, and the *streptococcus*—has been already considered. For a further discussion of the influence of tuberculosis see under "Diagnosis," p. 139.

Age and Sex.—Pleurisy is common at all ages. It is not rare in infancy, common in adolescence, attains its maximum frequency in early adult life, and continues somewhat frequent in old age. Of patients dying at advanced ages of various diseases it is the exception to find the pleuræ free from morbid changes. At the Royal Victoria Hospital, Belfast, in the decade 1891-1900, of a total of 9629 admissions 304 were cases of pleurisy, a proportion of 3·15 per cent. There is a preponderance—it is difficult to say to what extent—of cases in the male sex. Hospital statistics on this subject are apt to be fallacious.

Chill. — Considerable doubt surrounds the

question of the part played by chill in the causation of pleurisy. The writer has inquired into the subject in 74 cases which have been under his observation, chiefly in hospital practice. In 26 a history of chill was obtained, while in the remaining 48 cases there was no such history. In most cases chill took the form either of a wetting or of a rapid cooling of the body when overheated. When it is remembered with what facility a history of chill is volunteered, especially by patients of the hospital class, perhaps the noteworthy fact about these figures is not that a history of chill was available in one-third of the cases, but rather the absence of such a history in two-thirds. Still, the influence of chill cannot be eliminated. Those who hold that all cases of pleurisy are due to the presence of micro-organisms regard chill simply as a predisposing cause, as "lowering the vitality" of the organism, and so rendering it more susceptible to microbic invasion. As regards the influence of chill and its possible relation to the action of bacteria, pleurisy stands on much the same footing as pneumonia.

Specific Fevers.—Pleurisy is a not uncommon complication of some of the specific fevers, especially scarlet fever and smallpox. It is somewhat unusual in typhoid fever. Influenza is frequently assigned as a cause—often erroneously.

Bright's Disease.—In all forms of chronic Bright's disease we find a proclivity to inflammations of the various serous membranes, and the pleuræ are frequently affected.

Rheumatism.—It seems doubtful if there is any real causal connection between rheumatism and pleurisy. Of the 74 cases in the writer's practice, rheumatism was noted as being present only in 6. In the case of two diseases which are both sometimes associated with chill this proportion does not seem to exceed the possible limits of accidental concomitance. Valvular disease of the heart was noted in only 4 out of the 74 cases, a fact which tends to corroborate the view that there is no definite relation between pleurisy and rheumatism. It would appear that in the present state of our knowledge there is not sufficient ground for postulating the existence of a "rheumatic pleurisy."

Clinical History. — *Onset.* — In a small number of cases the onset of the disease is acute and "pneumonic" in type. There are grounds for thinking that these cases are due to *pneumococcus* invasion. In a larger group of cases, perhaps about one-half, the onset, though less acute and severe than in the above class, is well marked. The writer found this mode of onset in 18 out of 39 cases. In a third group of cases the onset is gradual but fairly definite, while in a fourth group the onset is entirely latent, and the disease escapes detection until the patient seeks advice for weakness or shortness of breath, and a pleural effusion of some duration is discovered. The first symptom is usually "stitch" in the side, with embarrassed breathing and some rise of temperature. A series of "chills" is common; a single severe rigor is rare. Vomiting may occur, and in children convulsions are sometimes noted. The temperature rises irregularly, and does not usually exceed 102° to 103° F. In some cases fever is absent. The course and duration of the pyrexia present much variation. The aspect and decubitus of a patient in the early stage of acute pleurisy are often characteristic. He lies on his back, or on the sound side, often curled up in a constrained position in the bed, and he leans forward and presses his hand to the affected side. His face shows that his suffering is severe. In addition to "stitch" in the side there may be a "referred pain" in various parts of the thorax or abdomen. The breathing is shallow and ineffectual, but it is evident that the patient refrains from taking deep breaths not from inability to do so, but simply through fear of exciting pain. There is rarely any of the acute dyspnœa, the *besoin de respirer*, which is so marked in pneumonia. The face is slightly flushed, and in rare cases herpes may be present about the mouth. There are the usual symptoms characteristic of pyrexia.

In a short time, usually within forty-eight hours, the patient's condition undergoes a change. Effusion sets in, the pain in the side is relieved, and the patient feels better, but is conscious of increasing shortness of breath, especially if he makes any exertion. He now finds himself more comfortable lying on the affected side, as this allows the lung on the sound side free play. The temperature remains usually at 101° to 103° F. in the evenings, with morning remission. The patient's condition may now undergo only slight change for some days, the state of the respiratory function depending largely on the amount of the effusion. In favourable cases absorption sets in towards the end of the second or the beginning of the third week, and may proceed rapidly. Pain in the side commonly reappears at this stage. Convalescence may be complete at the end of three or four weeks, but is often delayed. Cough is usually present throughout the disease, but is sometimes absent. It is often hard and unsatisfying, and accompanied by only scanty expectoration, which may be blood-stained at the outset; later it is usually mucoid or mucopurulent. Where œdema of the lung has supervened, on *paracentesis thoracis*, the expectoration is profuse and watery. Fœtor of the expectoration usually indicates that a fœtid empyema has burst through the lung. Diarrhœa is common. The urine is febrile, and not infrequently contains a little albumin. Attacks of pleurisy present almost every grade of severity, and the symptoms vary accordingly. A tedious convalescence may occur, even in cases which

ultimately make a good recovery. If the temperature reach 104° F., or the respirations exceed forty per minute, it is probable that pneumonia is present. A hectic temperature and profuse sweating may be present in cases where the effusion consists of limpid serum. Symptoms cannot be relied upon to determine the nature of the effusion (see under "Empyema," p. 144).

In the so-called "latent" or "quiet" pleurisy symptoms are sometimes said to be absent, but evidence of some degree of weakness and shortness of breath can always be elicited.

Physical Signs.—The characteristic sign of pleurisy in the first stage, before effusion has set in, is friction, which may, however, be absent. This is a scraping, grating, or creaking sound of variable, but usually considerable, intensity. Sometimes it has a greasy or sticky quality, and may be difficult to distinguish from crepitus (see under "Diagnosis"). It is usually heard both with inspiration and expiration, but is often loudest towards the end of inspiration. It gives the impression to the ear of being "superficial." It ceases when the breath is held. It is not affected by coughing. It may be intensified by pressure of the stethoscope. Pleural friction is most often heard in the inferior antero-lateral region of the chest or in the axilla. It is also common at the base of the lung posteriorly. If it be audible at the apex the suspicion of tuberculosis is strengthened. It is often confined to a very limited area, but is sometimes heard somewhat widely over the affected side. Friction fremitus is common. While there is at this stage no true flattening of the affected side, the patient expands it as little as possible owing to the pain. The lymph present on the surface of the pleura at this stage does not cause any recognisable dulness on percussion, nor any auscultatory phenomena other than friction.

Signs of Pleural Effusion.—Slight effusions may escape detection, but the presence of any considerable degree of effusion gives rise to a characteristic series of physical signs.

(*a*) *Inspection.*—The affected side is more or less immobile, either in its entire extent in very large effusions, or in a variable proportion of its inferior area. The antero-posterior diameter of the side is increased. The over-action of the healthy side is often marked. The intercostal spaces are usually, but by no means always, flattened. In recent effusions of moderate extent the normal dimpling of the spaces may be preserved for some time. Bulging of the intercostal spaces is excessively rare.

(*b*) *Palpation.*—In the great majority of cases vocal fremitus is either diminished or lost. In some cases, especially in women and in debilitated patients, the normal vocal fremitus is so weak that the interpretation of this sign becomes difficult, but apart from such cases the decided diminution or loss of vocal fremitus is one of the most trustworthy signs of the presence of fluid in the pleural cavity. It may, however, be fallacious (see under "Diagnosis," p. 139).

(*c*) *Percussion.*—Increased sense of resistance and dulness co-extensive with the area of the fluid are practically invariable. According to Guttmann dulness only becomes appreciable when the layer of fluid has a thickness of at least $1\frac{1}{2}$ cm. Probably few physicians would now accept Skoda's dictum that "the percussion sound is not altered by mere thickening of the pleura, nor by layers (several lines deep) of fluid or solid exudations upon it." The dulness is not complete until the lung is entirely emptied of air by the pressure of the fluid. In large effusions the dulness takes on a special "wooden" quality, which may be distinguished by the ear from the dulness of consolidation. The dulness usually is first manifest at the base of the lung posteriorly, and in small effusions it may be confined to this region. With an increasing effusion it invades the lateral region and the front, rising to a variable level, and in some cases occupying the entire side. In a considerable proportion of cases the upper level of the fluid is a curved line, the highest point of the curve being often in the axilla (Ellis and Garland). In patients who have been long bedridden the highest level of the fluid is usually found posteriorly. The outline of the dull area may be variously conditioned by recent or old adhesions, and may present many variations, which may suggest difficulties in diagnosis. It is quite exceptional for the level of the fluid to be altered by change of posture on the part of the patient. In a doubtful case this test is usually valueless. In the case of a considerable effusion which does not fill the entire pleural cavity the percussion note below the clavicle on the affected side is often tympanitic. This interesting fact was first observed by Skoda, and hence the sign has been called "Skodaic resonance." Its explanation is doubtful. Skoda's theory was that "when the lung is much reduced in volume by compression, but still contains air, its sound is invariably tympanitic." C. J. B. Williams thought that the tympanitic note was produced in the trachea and the large bronchi, a view which is not, perhaps, very probable.

(*d*) *Auscultation.*—Enfeeblement or obliteration of the normal vesicular murmur, and weakening, with some alteration of tone of the normal vocal resonance, are the rule, but the auscultatory signs of pleural effusion present great variations, and require to be interpreted with caution. Bronchial breathing is common, especially in children, and may either gradually give way to more or less complete silence, or may continue during the stage of effusion. In the writer's experience the bronchial breathing

of pleural effusion has usually had a more or less "distant" quality, which may help to distinguish it from the bronchial breathing of consolidation, but this rule is not invariable. It is probable that where bronchial breathing is present in pleural effusion the large bronchi are still patent.

Vocal resonance presents considerable variations, and should not be much relied upon in diagnosis. In the writer's experience one of the most usual changes is a certain flatness or loss of tone, rather than a mere weakening of the voice on the affected side. Sometimes the vocal resonance is simply weak, sometimes there is bronchophony. The term ægophony has been applied to a certain bleating quality of voice occasionally heard near the angle of the scapula at the upper level of moderate effusions. This sign is rare. It is of small diagnostic importance, and the term "ægophony" might well be dropped.

Baccelli thinks that the whispered voice is well conveyed through serous, but not through purulent effusions (Baccelli's sign). This observation has not been generally confirmed.

(*e*) *Mensuration.*—In large effusions the semi-circumference of the chest is increased on the affected side, but, as Gee well points out, "by the passage of the elliptical form into the circular, considerable increase in the sectional area of the chest may occur whilst the length of the periphery remains the same. Moreover, the displacement of the mediastinum thrusts the heart into the unaffected side. Add this consideration, too, that the walls of the healthy side must follow the antero-posterior projection of the diseased side, and then it will be plain why, as a matter of fact, the perimeter of the affected side often measures very little more, nay, sometimes even less, than the side which is not diseased." The cyrtometer may be used with advantage.

(*f*) *Succussion.*—The venerable sign, "Hippocratic succussion," one of the earliest fruits of medical observation, is, of course, only present when the pleura contains air as well as fluid.

(*g*) *Displacement of Organs.*—Displacement of the heart is the rule, displacement of the liver and spleen the exception, in pleural effusion. The displacement of the heart is more marked in left than in right effusions. The writer has removed 45 oz. of serum from the right pleura in a patient in whom no displacement of the heart could be made out. Such exceptional cases are, however, rare, and great importance must be attached to the position of this organ in any case of doubt or difficulty. Neglect of this rule has been a fruitful source of errors in diagnosis. The usual limits of displacement of the heart are the right mammary line and the left axillary line, but these limits may be exceeded. In addition to lateral displacement the heart is often raised. Under these circumstances the impulse may be due either to the normal apex or to the right ventricle. Epigastric pulsation may occur.

A systolic murmur is often audible over the heart in pleural effusion, and is probably due to some altered relation between the heart and the great vessels. It disappears with absorption of the effusion.

Upon the unaffected side the movements are exaggerated, and the vesicular murmur is intensified.

Stage of Absorption.—After a period varying from two or three days to two or three weeks, serous effusions in a large majority of cases begin to undergo absorption. The chief signs of the receding wave are the reappearance of vocal fremitus, if this has been entirely absent, some diminution in the absolutely wooden tone of the percussion note, the gradual return of displaced organs to their normal position, and the reappearance of friction sound and of the vesicular murmur. According to Gee the liquid disappears in something like the following order:—From the vertebral groove near the root of the lung; from the supra-mammary region; from the rest of the vertebral groove and infra-scapular region; from the infra-mammary region; and, lastly, from the lower lateral region. He adds, however, that "we must be prepared to find the residue of liquid in almost any part of the chest." In the writer's experience the fluid has often lingered longest at the base of the lung posteriorly, and this is probably the rule in patients who have been much confined to bed.

The rapidity with which absorption is accomplished varies within very wide limits. Sometimes it is fully accomplished in a few days, more often it is somewhat delayed, and not uncommonly it is a tedious and lingering process. Slight dulness on percussion and some impairment of the vesicular murmur may remain for long periods, or for life, in patients who have apparently made a complete recovery. In many cases, however, no trace of the attack remains. The notion that in this latter group of cases permanent adhesions have occurred does not seem warranted by the evidence at present available to us. More or less retraction of the side often remains after an attack of pleurisy. The mythical cases of "people living with one lung" are usually instances of old pleurisies where an extreme degree of retraction of one side of the chest has occurred. In other cases the lung undergoes more or less carnification, and the patient remains permanently short-winded and debilitated, or becomes the subject of tuberculosis.

Complications.—Pleurisy has few characteristic complications. The other serous membranes—most often the pericardium, very rarely the peritoneum—may share in the morbid process. Albuminuria is not uncommon, but is

sometimes merely febrile, sometimes it points to a pre-existing nephritis, of which pleurisy is a frequent concomitant. In chronic cases dilatation of the heart and dropsy are not infrequent. Clubbing at the ends of the fingers is common in empyema, but not unknown in chronic cases of serous effusion. Pneumothorax is related rather to empyema than to serous pleurisy (see "Pneumothorax," p. 145). Acute œdema of the lungs occasionally, but very rarely, supervenes upon aspiration.

Differential Diagnosis.—The diagnosis of pleurisy is usually an easy task. Sometimes, however, there may be difficulties, and occasionally a positive opinion may be impossible. Difficulties may arise either (*a*) in connection with pleural friction, or (*b*) in connection with the recognition of effusion. We shall consider the subject under these two heads.

Pleural friction is in a large majority of cases unmistakable. Its rubbing or scraping quality, its superficiality, its dependence on the respiratory act, its independence of coughing, its association with pain, its intensification by pressure of the stethoscope, its usual distribution, make its recognition generally obvious. Its distinction from pericardial friction rarely gives rise to serious difficulty. Pericardial friction is much more rapid than pleural friction, it has usually a to-and-fro quality, it is confined to the præcordial area, and is most frequent at the base of the heart, it does not cease, except in very rare cases, when the breath is held. It must be borne in mind that the reflection of the pleura over the pericardium may be affected, and may give rise to confusing physical signs. A much more serious difficulty arises sometimes in connection with some cases of sticky and greasy exudation on the pleural surface, which may cause a sound readily mistaken for crepitus. The following points will help in obviating this error. Friction is "superficial," crepitus is deep. Friction is not affected by coughing, crepitus often is affected by coughing. Friction is often intensified by pressure of the stethoscope, crepitus never. Friction is often audible throughout the greater part of inspiration and expiration, though there are exceptions to this rule; crepitus is often, though by no means always, confined to the end of inspiration and the beginning of expiration. Friction is commonly accompanied by pain, crepitus rarely. Lastly, if we examine in the neighbourhood of the doubtful sound we shall often detect unambiguous friction or crepitus, which may settle the diagnosis.

Pleurodynia may be confused with pleurisy. Many cases diagnosed as pleurodynia are really cases of "dry" pleurisy. Error may be best avoided by careful physical examination, and by the use of the thermometer; the presence of pyrexia to be allowed much, its absence little, weight.

More formidable are the difficulties which sometimes beset the diagnosis of pleural effusion. Most observers are agreed that in exceptional cases, especially in children, the signs of pleural effusion may be indistinguishable from those of consolidation. As an abstract proposition this is unquestionably true, yet the cases in which the distinction is impossible are not numerous. Too much reliance upon the auscultatory signs is the chief source of error. We should rely chiefly on (1) the state of the vocal fremitus, viz. upon its diminution or disappearance in pleural effusion, its normal or increased intensity in consolidation; (2) the outline and limits of the dull area, sometimes characteristic in pleural effusion (see *ante*, "Physical Signs," p. 137); and (3) the displacement of organs, especially the heart. If a careful inquiry under these heads still leaves the case in doubt, an exploratory puncture should be made in all cases in which a positive diagnosis is desirable.

In some cases of pneumonia a temporary blocking of the bronchial tubes with secretion may cause signs which simulate those of pleural effusion, viz. loss of vocal fremitus, loss of breath sounds, and diminution or altered quality of vocal resonance. The effect of coughing and expectorating will serve to clear these cases up.

Malignant disease of the lung may give rise to signs which cannot be discriminated with certainty from those of pleural effusion. In a recent case in the writer's practice the lower portion of the right lung presented the signs of wooden dulness on percussion, complete absence of vocal fremitus, and entire loss of breath sounds. The presence of some suspicious superficial nodules and the general history of the case gave rise to the suspicion of malignancy, and at the autopsy the right lung was found to be invaded by a large mass of encephaloid carcinoma. In such cases, which are of extreme rarity, reliance must be placed, not on physical signs, but upon the history and symptoms. Persistent pain is usually present in malignant disease of the lungs, hæmoptysis is common, but not invariable, the general progress of the case is characteristic of malignancy, and pressure signs tend to develop, but may be late in appearance.

Diaphragmatic pleurisy may give rise to difficulty. Severe pain is the chief feature of these cases at an early stage, more or less pyrexia will probably be present, and after a short time clear physical signs will tend to appear, as the pleurisy is probably never wholly confined to the reflection of the pleura upon the diaphragm.

Encysted or loculated effusions, usually conditioned by the pressure of old adhesions, may give rise to puzzling physical signs. Loculation may occur at any part of the pleural cavity, but seems to be most frequent in the lower lateral or posterior region. It may occur between the lobes of the lung. Puncture should

be resorted to in these cases, and it is important that the trochar should be inserted at a point where the signs of the presence of fluid are well marked. Otherwise the failure to find fluid may unjustly discredit the diagnosis.

Collapse of the lung, acute or chronic, closely simulates pleural effusion, but the history of these cases is different from that of pleurisy, the side is retracted, the intercostal spaces are not flattened, and the heart is not displaced towards the sound side. Attention to these points may be sufficient to prevent error, but if necessary puncture should be resorted to.

The possibility of subphrenic abscess should be borne in mind in cases which suggest pleurisy, but present abnormal physical signs (see under "Subphrenic Abscess," vol. ii. p. 343 *et seq.*).

The general question of the relation of pleurisy and tuberculosis may be here conveniently considered. The pathological aspect of the subject has been already dealt with. It remains to consider its clinical bearings.

Vincent Bowditch of Boston found that of 91 cases of pleurisy occurring in the practice of his father, Henry J. Bowditch, between the years 1849 and 1879, 32 died of phthisis, or were certainly tuberculous. Fiedler found that of 92 cases of pleurisy with sero-fibrinous exudation, 28 cases were dead of pulmonary tuberculosis within two years, while in many of the remaining cases the presence of tuberculosis was suspected or probable. Barrs of Leeds found that of 62 cases of acute pleurisy occurring at the Leeds Infirmary 22 were dead of phthisis within six years. After an exhaustive treatment of the whole subject, Kingston Fowler concludes that "the bacillus of tubercle is the causative agent in a very large proportion of cases of sero-fibrinous pleurisy, and that this is true when the attack is apparently simple, idiopathic, and attributed to a chill." The same writer further points out: "It must be borne in mind that the course of tuberculosis of the serous membranes is far more favourable than when the disease attacks the lungs and other organs. Of this the clinical history of tuberculous peritonitis affords a striking example. The inference that in many cases of tubercular pleurisy the disease is arrested at the time and never reappears is therefore justified, and is supported by pathological experience." We have seen that Germain Sée regarded all cases of acute pleurisy as tubercular, and that Landouzy from the side of pathology takes practically the same view.

While there is no longer any doubt that the association of pleurisy and tuberculosis is much more intimate than was formerly suspected, there are some considerations which must make us pause before accepting the extreme view on this subject which is now current, especially in France. The onset in pleurisy is sudden in about one-half the total cases. In tuberculosis a sudden onset is the rare exception. Pleurisy following a definite chill, though much less common than was formerly thought, is nevertheless somewhat frequent. Chill does not produce tuberculosis. The incidence of pleurisy and tuberculosis with regard to age and sex shows some points of apparent contrast, but the subject requires further elucidation. Pleurisy most often appears to begin in the lower antero-lateral region of the chest—*i.e.* in that part of the side least protected by muscle—and shows none of the preference for the apical region which is so marked in pulmonary tuberculosis. The course of the temperature in pleurisy is unlike that of tuberculosis. The general progress of the disease in pleurisy and the early and favourable convalescence of the large majority of cases are unlike tuberculosis. It will be admitted that the clinical course of acute pleurisy bears only a distant resemblance to (*e.g.*) that of tubercular peritonitis. While these points must be borne in mind before we can formulate a definite conclusion on this most important and difficult subject, probably most physicians will agree with Osler when he says: "I confess that the more carefully I have studied the question the larger does the proportion appear to be of primary pleurisies of tuberculous origin." It has been seen that the *bacillus tuberculosis* is very rarely present in serous effusions, and the theory that all "sterile" effusions are tubercular is still only an hypothesis. Much will depend upon the results of further inoculation experiments. If it can be conclusively proved that inoculation with serous pleuritic fluid produces tuberculosis in a large and fairly constant proportion of cases the conclusion to be drawn will be obvious. It should not be forgotten that the fact of a patient who has recovered from an acute pleurisy subsequently developing tuberculosis does not prove that the pleurisy was tuberculous. At the most it only raises a presumption that such may have been the case. But it is at least arguable that in some of these cases—perhaps a considerable proportion—a non-tuberculous pleurisy, not thoroughly recovered from, may have weakened the resisting power of the lung, and so rendered it more susceptible to invasion by the bacillus of tubercle. Such a sequence of events is entirely consonant with our present knowledge of the causation of pulmonary tuberculosis.

If pleurisy occur in a patient whose general health is from any cause debilitated, or whose history suggests the probability of antecedent pulmonary mischief, the suspicion of tuberculosis will be much strengthened. In such cases the sputum, if obtainable, should of course be examined with special care.

Prognosis.—The mortality during the acute

stage of pleurisy is insignificant. In 39 consecutive cases in the writer's practice death took place in two instances: in one from meningitis, in the other from heart failure consequent on delirium tremens. In neither case did the fatal issue seem to be directly dependent upon disease of the pleura.

Pleurisy derives its great importance almost entirely from three considerations, viz.: (1) it may lead to empyema or chronic serous effusion; (2) it may permanently damage the lung and lead to prolonged or permanent ill-health; (3) it may be either a manifestation or a cause of tuberculosis. Our anxiety with regard to patients suffering from pleurisy is chiefly concerned with the possibility of a lingering and unsatisfactory convalescence and of sequential disease. It is important to bear in mind that, on the one hand, tedious and troublesome cases sometimes ultimately make an excellent recovery, and, on the other hand, that a rapid convalescence does not exclude the possibility of a later development of tuberculosis.

Sudden death in the course of acute pleurisy is not unknown, and seems to be dependent upon thrombosis of the right side of the heart.

Pericarditis is a serious complication.

Treatment.—The general rules applicable to the pyrexial state should be observed. In the early stage the patient's chief complaint is of pain, which is usually severe. A hypodermic injection of morphia, repeated if necessary, and followed by a few doses of Dover's powder, affords a safe and efficient means of relief. The abstraction of blood by leeching, wet cupping, or venesection is very rarely necessary. The respiratory movements being intensely painful, much relief will be obtained by tightly bandaging the side, or by the application of strips of plaster, after the manner recommended by F. T. Roberts. Poultices of linseed and mustard, or hot fomentations combined with laudanum, are often grateful to the patient. The bowels should be moderately opened and a febrifuge mixture administered. The diet should be liquid. The above measures usually suffice until the effusion has reached its height. In a large proportion of cases the effusion is spontaneously absorbed, and active treatment is both unnecessary and useless. The bowels should be kept open without active purgation, and a mixture containing acetate of potash, spirit of nitrous ether, and digitalis, will assist excretion by the skin and kidneys. The value of the time-honoured blister of cantharides or iodine is very doubtful. Our chief concern at this stage is to watch for the normal recession of the tide of effusion. As the pyrexia abates the writer is in the habit of substituting a mixture containing 2 or 3 grains of iodide of potassium combined with 3 or 4 grains of the citrate of iron and ammonia *pro dosi* for the diaphoretic and diuretic mixture already mentioned. If the effusion soon begins to show signs of subsidence a general tonic line of treatment should be early undertaken, bearing in mind the dangers of a lingering convalescence or of the development of tuberculosis. If the effusion persists at its highest level the question of more active treatment must be considered. We have to choose between such measures as active purgation, blistering, and the limitation of fluids on the one hand, and aspiration on the other. The writer has seen little success from the former methods, while the results of aspiration are often more satisfactory. Osler, whose opinion must carry great weight, states that he has seen large effusions disappear rapidly under a dry diet and full doses of Epsom salts. These methods may have a trial, but if no signs of absorption appear in the course of the third week aspiration should be performed if there be no contra-indications. The question arises whether we are justified in waiting so long before resorting to this expedient. It is probable that we are justified for the following reasons:—In the course of the first two weeks spontaneous absorption is always probable. The dyspnœa consequent on the exudation is rarely so urgent as to demand relief at an earlier stage, but urgent dyspnœa should be regarded as an indication for aspiration at any stage of the disease. It is remarkable, however, how soon patients get accustomed to the effusion and how little they suffer from dyspnœa, so long as they make little or no exertion. Further, early aspiration seems to increase the tendency to a fresh accumulation of fluid, and it does not seem to prevent deformity of the chest. One of the worst cases of falling in of the side ever observed by the writer occurred in a child in whom, owing to somewhat urgent dyspnœa, it was thought advisable to perform aspiration about the seventh or eighth day of the disease. Large effusions, as a rule, absorb tardily, and in most of these cases aspiration will be required. Aspiration is also indicated in the rare cases in which the effusion is bilateral. As regards the proportion of cases in which aspiration will be called for no rule can be laid down. In one series in the writer's practice aspiration was performed in 13 out of 39 cases. He has never had reason to regret the performance of the operation, but has sometimes regretted that for some reason or another the operation was postponed or omitted. The effect of aspiration varies. In exceptional cases it is followed by rapid convalescence, and no further treatment is required. The writer can recall a case of a young girl whose left pleura had been allowed to remain full of fluid for eight or ten weeks, and who rapidly convalesced after a single aspiration. Such results are, however, exceptional. Much more often the fluid reaccumulates, and the operation has to be repeated. The effect of aspiration upon the

temperature is variable. In 4 of the 13 cases already mentioned aspiration was followed by a marked fall in temperature. In several cases aspiration had no effect upon the temperature, and in one case the pyrexia seemed to be aggravated by the operation. The idea that persistent pyrexia contra-indicates aspiration seems to be without foundation.

The management of the convalescent stage of pleurisy is a matter of the utmost consequence, to which hardly sufficient attention is usually paid. We shall first consider the management of this stage in cases which pursue a fairly normal and favourable course, and, secondly, in those troublesome and difficult cases of recurring and persistent effusion. In the former cases a definitely tonic line of treatment should be early adopted. When the pyrexia has disappeared or becomes trifling, the patient should leave his bed, take gentle exercise at first indoors, and before long in the fresh air, and be instructed to breathe deeply and use the dumbbells. A change of air—preferably to upland air—and gentle daily walking exercise on moderate inclines will often have the happiest results in these cases. Singing may be practised with advantage. The diet should be as liberal as the state of the digestion will permit. The possibility of subsequent phthisis is always to be borne in mind, and a winter at one of the Alpine stations will sometimes be advisable. It is reasonable to hope that by such measures the proportion of these cases which ultimately become phthisical may be materially reduced. Much more difficult is the management of those cases, by no means rare, where in spite of repeated aspiration the fluid obstinately reaccumulates, and no real progress is made. Aspiration should have a fair trial, but no rule can be laid down as to how often it should be repeated. The writer is averse to very frequent repetition of the operation, except as a palliative measure in obviously incurable cases. It seems to be generally agreed that free incision and drainage is not to be advised in these cases, and that injections of astringents or antiseptics are useless. The writer has no experience of these methods. Perhaps the best course to pursue in these unpromising cases is to adopt some approximation to the tonic and hygienic line of treatment advised for the more favourable class. At best the outlook is gloomy. Too often the heart becomes dilated, dropsy sets in, or else an intractable empyema is set up, or in some other way the general health is ruined. Lardaceous disease sometimes marks the final stage. Probably a large proportion of these cases are tubercular.

In dealing with any case of pulmonary disease the fact of an antecedent pleurisy should always be allowed considerable weight. It suggests caution in prognosis and a careful search for evidence of tuberculosis. It should not be forgotten in connection with life assurance forecasts.

(For a discussion of the surgical aspects of aspiration, see "Paracentesis Thoracis," p. 150.)

Pleurisy in Children

"Pleurisy in children," says Henoch, "differs in no essential particular from the same disease in later life." This is true, there being no features of the disease peculiar to any period of life, yet pleurisy in the child requires special attention. The difficulties of diagnosis are greater in the child than in the adult, and misleading signs and symptoms are more common. We shall briefly summarise the more important points.

(*a*) *Mode of Onset.*—Vomiting, epileptiform convulsions, and other symptoms of cerebral irritation are commoner in the child than in the adult, but, on the other hand, an insidious onset is very common in the child, and the disease can be recognised only by physical examination.

(*b*) *Nature of the Effusion.*—The effusion is more often purulent in the child than in the adult. Goodheart found pus in 78 out of 149 cases of pleural effusion in childhood. It is about an even chance whether we shall find pus in any case of acute pleurisy with effusion occurring in a child. Loculation of the fluid is common.

(*c*) *Physical Signs.*—The peculiarities of the physical signs of pleurisy in the child depend upon the facts that at this age the chest walls are more yielding, and the lung tissue more elastic and less easily compressed than in the adult. Deep percussion will often yield resonance, even in the presence of a large effusion. Light percussion is more satisfactory. Skodaic resonance is well marked. Alterations in the tactile fremitus are less trustworthy in the child than in the adult.

Bronchial breathing is the rule in childhood, either early in the case or lasting throughout the entire effusion stage. It is often but not always weak and "distant" in quality. Sometimes the breathing has the normal vesicular character, but is less distinct than in health. "Good though deficient vesicular murmur," says Goodheart, "may be present all over the side which is full of fluid, and unless this is remembered there is likely to be a mistake in diagnosis." In certain cases of pleurisy in children the signs, especially in the mammary region, may closely simulate those of cavity. The breathing is hollow, and there are adventitious sounds not easily distinguishable from gurgling râles. In such cases the general history of the case, the examination of the sputum, the position of the heart, and the result of puncture may be relied upon to establish the diagnosis. The exaggeration of the breath sounds on the healthy side is marked.

(*d*) *General Considerations.*—Pleurisy is more often a sequel of the acute infective diseases in the child than in the adult. Pericarditis is said to be commoner in early life than at a later period. The course and results of pleurisy do not present any striking peculiarities in childhood. Rapid absorption is common, but so also is a lingering and tedious convalescence. Marked deformity of the chest may remain after an attack, but often undergoes much subsequent amelioration. In empyema the prospects of cure by simple aspiration are much better in the child than in the adult, but incision and drainage will usually be necessary.

Varieties of Pleurisy. — No satisfactory classification of pleurisies is at present possible. Most of the usual varieties have been incidentally alluded to in the course of the foregoing description. The subject can only be very briefly considered here.

Most of the classifications which have been adopted involve unverifiable hypotheses, or seek to establish distinctions which a fuller knowledge may show to be invalid. Enough has been said to show that the differentiation of tubercular pleurisy from other varieties, which is the great practical desideratum, cannot be established with certainty either by clinical or pathological evidence. "Latent" or "quiet" pleurisy has no claim to be regarded as a separate entity, nor can "diaphragmatic pleurisy" be erected into a distinct variety. It is very questionable if pleurisy ever remains limited to the reflection of the pleura upon the diaphragm (*see* vol. ii. p. 339, "Diaphragm"). Three types of pleurisy remain for consideration which may fairly demand separate consideration, viz. dry pleurisy, and chronic pleurisy and empyema.

Dry Pleurisy.—In this condition the pleural surface is covered in part with one or more layers of lymph, and effusion does not take place. The symptoms are pain in the side, slight pyrexia, which may, however, be absent, and some degree of general malaise. Friction is heard on examination of the chest. It is commonly posterior or antero-lateral in situation, usually in the neighbourhood of the base of the lung, and is most often quite limited in extent, but in exceptional cases may be heard almost over the entire side of the chest. The course of these cases is often tedious, but ultimately adhesions usually form between the opposing pleural surfaces, and recovery takes place.

The same doubt overhangs the ætiology and significance of these cases which appertains to the subject of acute pleurisy. Whether a "dry" pleurisy is ever the result of simple chill, whether it ever arises in an individual otherwise quite healthy, whether the presence of this condition warrants a suspicion of tuberculosis, whether the after history of these cases shows that their ultimate course is unfavourable—all these questions are at present unsettled. The fact that in almost every patient dying of a chronic malady some adhesions of the pleuræ are found, points to the conclusion that this membrane readily undergoes plastic exudation in patients whose general condition is bad, or who are the subjects of some constitutional malady. To infer that the pleural adhesions so constantly found in the dead-house are always, or even in the majority of cases, tubercular, seems a large and unverifiable assumption. Direct proof of their tubercular nature is usually wanting. On the other hand, it seems doubtful whether a "dry" pleurisy ever arises as the result of simple chill in an individual otherwise quite healthy, and a pleurisy of this type should be watched with as much, or even greater care than an ordinary acute pleurisy. Where friction is limited to the apical region the suspicion of tuberculosis will be much strengthened.

The treatment consists in the relief of pain, general tonic measures, and watchfulness regarding possible developments. The subject of "dry" pleurisy should, if possible, shun indoor occupations, and all depressing conditions of life and work should be reduced to a minimum. The sputum, if obtainable, and the urine should be carefully examined. It must not be forgotten that in exceptional cases "dry" pleurisy takes on an acute development, and effusion may suddenly set in.

Chronic Pleurisy. — This subject has been already incidentally considered. The main facts may be here conveniently summarised. Chronic pleurisy may be a sequel of the acute or subacute affection, or the attack may be chronic throughout. It may be attended by moderate or large effusion, or may belong to the type of the so-called "dry" pleurisies. The so-called "latent" or "quiet" pleurisy already described, in which a large effusion collects without definite symptoms, is one variety of chronic pleurisy with effusion. In another class of cases an ordinary attack of acute or subacute pleurisy is characterised by persistence of the effusion, or by its repeated recurrence after aspiration. A large proportion of these cases are probably tubercular. In a third type of case the exudation is plastic, more or less extensive adhesion of the pleural surfaces takes place, and the function of the lung is in some degree impaired. In very chronic cases thickening of the pleura to an extreme degree may occur, the newly formed tissue is often tough and unyielding in consistency, and sometimes it undergoes calcareous change. The lungs may undergo cirrhotic change. The general course and symptoms of chronic pleurisy present great variations. Amongst the most constant features are shortness of breath, cough, progressive debility, slight cyanosis, and diarrhœa. Pain is usually present, but may be slight, and may not be mentioned by the patient until inquired for by

the physician. At a late stage of some of these cases the symptoms of dilatation of the heart may complicate those proper to pleurisy.

The physical signs of chronic pleurisy depend upon the amount of the effusion, the degree of collapse of the lung on the affected side, and the consecutive changes in the chest wall. In some cases retraction of the side is a prominent feature, in others there is no retraction. Dulness on percussion, impairment of tactile fremitus, and feebleness of the respiratory murmur are usually present, but in very varying degree. In some cases friction is the only physical sign.

The prognosis is unfavourable as regards complete *restituo ad integrum*, but some of the less grave cases preserve a fair degree of health and fitness for work for long periods. It is not well to take a grave view of slight cases of chronic pleurisy, but the possibility of tuberculosis must always be borne in mind. Cases of persistently recurring large effusions are very unfavourable. The treatment has been already considered (see p. 141).

Empyema

The bacteriology of empyema has been already considered. The organisms most often found are the pneumococcus, the streptococcus, the staphylococcus, and various bacteria of putrefaction. The bacillus tuberculosis is, on the whole, rare. There is a growing consensus of opinion amongst bacteriologists that a pneumococcus empyema is the most favourable variety, and a streptococcus empyema the least favourable. The significance of the presence of the bacillus tuberculosis must be estimated on general principles.

Ætiology.—In a large proportion of empyemata the effusion is purulent from the onset, especially in children, and due either to some intensity of the morbid process or to the weak resisting power of the individual. The idea that serous pleurisies often become purulent as the result of repeated aspiration rests on no secure evidence. If, however, on a first aspiration the fluid is somewhat turbid in appearance and is found to be rich in cellular elements, empyema will probably result. These cases are from the outset on the way to become purulent.

Pneumonia is one of the most important causes of empyema, which may either accompany or succeed the pneumonic attack. In the former case the clinical course and the physical signs may present some puzzling features until the diagnosis is made out. The attack may set in with the ordinary signs and symptoms, and for a few days no special features may be present. But the crisis is delayed, the expected improvement does not take place, the looked-for crepitus redux does not appear, and gradually unequivocal signs of fluid in the pleura make their appearance. Puncture should be early practised in these cases, as a prompt diagnosis is imperative.

The pleurisy connected with scarlet fever and typhoid is often purulent.

Empyema may result from disease of bone, wounds and injuries of the thorax, malignant disease of the lungs or the œsophagus, rupture of an abscess or a tubercular vomica into the pleural cavity.

Tuberculous pleurisy is sometimes purulent, but is much more frequently sero-fibrinous.

Empyema may occur in association with septicæmia.

Pathology.—The effusion presents all gradations from a slightly turbid sero-pus to a thick, inspissated lumpy mass, the former condition being characteristic of recent, the latter of chronic cases. Often the effusion is of the consistence of cream. Fœtor is unusual, but may be found in traumatic cases, and in association with gangrene of the lungs or pleuræ.

Thickening of the pleura is usual, and may attain an extreme degree in very chronic cases. The fibrinous exudation becomes organised into granulation, and ultimately into connective tissue, and adhesions are formed. Loculation is common. The pus may become impregnated with calcareous salts, and calcareous matter may be deposited in thick layers upon the pleural surface.

Diagnosis.—There is only one means of diagnosing empyema, viz. by puncture and withdrawal of some of the fluid. Often, no doubt, the presence of pus may be correctly surmised, but neither by signs nor symptoms can the surmise be translated into certainty. The physical signs of empyema and those of serous effusion are identical and have been already fully considered. Nor will the symptoms suffice to afford a secure diagnosis. The writer has seen marked hectic fever, profuse perspiration, and wasting in cases where on aspiration the fluid proved to be limpid serum, and, on the other hand, has found pus where there was little or no pyrexia or other active symptom. Œdema of the side and clubbing of the fingers are equally fallacious. These signs point to the chronicity of the case, but shed no certain light upon the character of the effusion. The history will often give a hint, but it must be reaffirmed that until some of the fluid has been withdrawn the diagnosis remains simply a more or less probable guess. In making the exploratory puncture some precautions are necessary. The exploring syringe or trocar and cannula should not be too fine, otherwise the pus will not flow through it. The thickening of the pleura may be extreme, and great difficulty may arise in gaining access to the pleural cavity. Further, loculation is common, and the pus may be missed. This error may usually be

avoided by the operator taking care to insert the instrument at a point where the signs of the presence of fluid are unequivocal.

Treatment. — This is purely surgical (*see* "Surgical Section," p. 148).

Hydrothorax

Dropsy of the pleural cavity or serous effusion not due to primary changes in the pleura is a common condition, and is not unfrequently overlooked. It is common in heart disease, Bright's disease, or other chronic renal malady, and may be found in connection with aneurysm, thoracic tumours, chronic hepatic disease, and anæmia.

The nature of the effusion has been already considered. It is usually distinguishable from inflammatory or bacterial effusion by its lower specific gravity, its relative poorness in corpuscular elements, and its tardiness in coagulating.

The physical signs are not to be distinguished from those of ordinary pleural effusion, but friction is not present, and as the effusion is often double the heart may remain in its normal position. It is remarkable, however, how often the effusion is unilateral in cases where a bilateral effusion might be expected. A unilateral effusion is quite common in cardiac cases, perhaps more common than a double effusion. A very usual condition in these cases is to find a considerable effusion on one side, with a slight and barely recognisable effusion on the other. In renal disease the effusion is more often on both sides. Passive effusion into one or both pleural cavities is a very usual explanation of the aggravation of symptoms in a cardiac case, and should always be carefully borne in mind. In such patients, however, so long as they are confined to bed and making little or no exertion, a considerable degree of effusion may take place into one or both pleural cavities without any marked increase of dyspnœa.

The treatment of hydrothorax, apart from general medical measures, is aspiration, which should always be practised where there are any active symptoms. The prospects of spontaneous absorption are rarely good. They are, perhaps, best in cardiac cases.

Hæmothorax

An effusion of pure blood into the pleura probably occurs only as the result of trauma, or the rupture of an aneurysm, but effusions of more or less sanguineous serum (more rarely pus) are not very infrequent. The causes of this condition have been already indicated, viz. tuberculosis, malignant disease, the exanthemata, hæmophilia, scurvy, cirrhosis of the liver, and possibly inflammatory changes in profoundly debilitated subjects.

The physical signs do not present anything distinctive, but the associated conditions may give rise to a somewhat complicated clinical picture.

The chief interest of these cases lies in the prognostic significance of sanguineous effusion. This varies within wide limits, according to the ætiology, but for obvious reasons the prognosis is rarely favourable. A satisfactory issue, though not often to be anticipated, is by no means unknown. The writer has known comparative recovery with restoration of health for several years to follow a case of large sanguineous effusion in a young gentleman who was probably tubercular.

As regards treatment, aspiration is not to be lightly undertaken, but the propriety of the operation may be considered in cases where the amount of the fluid is causing serious inconvenience. Several cases are on record where the operation has been followed by recovery, but it may be attended by considerable risks. The presumption is, on the whole, against this procedure in hæmothorax.

Pneumothorax

Air in the pleural cavity is almost always associated with fluid—rarely serum, usually pus. That air is sometimes effused into the pleural cavity and subsequently absorbed without appreciable exudation is probable, but the clinical importance of these cases is slight. Practically pneumothorax and pyo-pneumothorax are almost convertible terms. In a large majority of cases pneumothorax is a consequence of tuberculosis.

Ætiology.—It is probable that pneumothorax is never a primary affection. Laennec believed the contrary, and held that it might arise by the exhalation of air from the pleura or by the decomposition of fluids in the pleural cavity. These views have not been confirmed by subsequent observers. Whether bacteriology will throw much new light on the ætiology of pneumothorax is still doubtful. E. Levy describes a bacillus which he has found in the exudate of pneumothorax, and to which he is disposed to attach importance. It is a short, thick bacillus with rounded ends, anaerobic, non-motile, and readily stained by aniline dyes. It occurs frequently in pairs, but often, also, in long chains or threads. Spore-formation was not observed to occur. At a temperature of 37° C. the growth of this bacillus proceeds actively, with a remarkable evolution of gas.

The two chief causes of pneumothorax are (1) the breaking down of a subpleural nodule in the course of pulmonary tuberculosis, and (2) the discharge of an empyema through the lung or the chest wall. Of remaining causes trauma and pulmonary gangrene are the least infrequent. Emphysema has been educed as a cause in a few instances, and inasmuch as bullæ containing air are common enough on the pleural surface in this disease, there is no antecedent improbability that pneumothorax might result. If these bullæ rupture it seems likely that the air is often reabsorbed, and that pneumothorax

does not as a rule ensue. In one case in the writer's practice pneumothorax was the indirect result of gastric ulcer, an abscess forming behind the stomach, which penetrated to the pleural cavity through the diaphragm, and ultimately discharged through the lung. Abscess of the lung, cancer of the lung, "apoplexy" of the lung, have been regarded as causes of pneumothorax. Such cases must be of extreme infrequency. The writer's experience is entirely in accord with the statement of Douglas Powell: "Excluding gunshot wounds, the discharge of empyemata, and gangrene of the lung, it is infinitely rare for pneumothorax to occur from any other cause than the breaking down of subpleural consolidations in phthisis."

The admission of air to the pleural cavity in consequence of the aspiration of an empyema is an occasional cause of pneumothorax.

As regards the relative frequency of the various causes of pneumothorax, pulmonary tuberculosis easily takes the first place. Saussier, whose statistics are quoted by Fraentzel, found pulmonary tuberculosis in 81 out of 131 cases of pneumothorax. These figures, as Fraentzel justly remarks, "evidently give a too small comparative proportion to pulmonary consumption." He regards this cause as present in about 14 out of every 15 cases. Walshe puts the proportion of cases coming under this head at 90 per cent. Empyema is a somewhat frequent cause of pneumothorax. Saussier found this cause present in 29 of his 131 cases. The other causes, excepting perhaps gangrene of the lung, are quite rare.

As regards the proportion of cases of pulmonary tuberculosis in which pneumothorax occurs, we find divergent views. Neil put it at 13 per cent, Williams at 10 per cent, West and Powell at 5 per cent. The writer's experience tends to corroborate the last figure.

Authorities differ as to which side is oftener affected, but the point is immaterial.

Pathology. — Most observers incline to the opinion that pneumothorax is most apt to occur in acute cases of pulmonary tuberculosis, especially in the acute caseating form, but Fraentzel holds the contrary view, and asserts that in his experience pneumothorax has arisen most often in chronic cases, and usually at a very advanced stage of the disease. This has also been the experience of the writer. Acute miliary tuberculosis and fibroid tuberculosis seem to have little tendency to cause pneumothorax. Violent fits of coughing and straining at stool seem to be the usual exciting causes of pneumothorax in tubercular cases. The tubercular masses break down near the surface of the lung, necrosis of the pleura occurs, and pneumothorax follows. If it were not for the formation of protecting pleural adhesions, pneumothorax would be a common, instead of a rare, phase of pulmonary tuberculosis.

There may be one or several openings, and the opening or openings may be large or small, patent and direct, or oblique and valvular. As regards the seat of the perforation, Williams found it in the upper lobe in 17 instances, in the middle lobe in 4, and in the lower lobe in 16, in the tubes between the upper and middle lobes in 1. Walshe was of opinion that "the pleura commonly gives way postero-laterally in the area comprised between the third and sixth ribs." West's figures give a large preponderance to the upper lobe.

The mode in which empyema or pulmonary gangrene gives rise to pneumothorax is too obvious to demand any detailed description.

The gas effused into the pleural cavity is found to have approximately the characters of expired air. J. Lasch states, on the authority of some unnamed observers, that by estimating the amount of oxygen and carbon dioxide in this gas, we can determine whether or not there is a free communication between the bronchi and the pleural cavity. If a free opening exists, the proportion of oxygen is over 10 per cent, that of carbon dioxide not exceeding 5 per cent, while if no such opening exists the proportion of oxygen is under 10 per cent, that of carbon dioxide over 10 per cent.

Douglas Powell estimated the state of the intrapleural pressure in 16 cases of pneumothorax. He found the pressure *nil* in 4 cases, and in 12 cases the pressure varied from $1\frac{3}{4}$ to 7 inches of water.

Clinical History.—*Mode of Onset.*—In a considerable proportion of cases the onset is sudden and well marked, and symptoms of severe shock are present. The patient feels a severe pain in the chest, he has the sensation "as if something had given way," dyspnœa becomes urgent, the pulse is much accelerated, and a clammy perspiration breaks out. Cyanosis is often a marked feature. The decubitus of the patient varies. Cough and expectoration sometimes cease temporarily.

It is by no means rare, however, to find these symptoms absent or ill marked. As Fraentzel truly observes, pneumothorax often comes on in advanced phthisis "without any particular complaint on the part of the patient, and without any decided aggravation of the previously existing dyspnœa; and it is only on a closer examination that we discover the presence of an accumulation of air in the pleural cavity." In cases where pneumothorax supervenes upon empyema the first indication of its presence may be the ejection of a large quantity of pus from the mouth. It is probable that the acuteness of the symptoms of onset has some relation to the functional activity of the lung on the affected side at the moment when perforation of the pleura occurs. If the lung be comparatively sound the symptoms will probably be urgent, while if it be extensively damaged by consolida-

tion, softening, and excavation the symptoms will tend to be more latent. According to Douglas Powell, the symptoms of pneumothorax "may be most closely simulated in an attack of acute pulmonary congestion supervening upon already advanced disease."

Progress of the Case.—The patient may die within a few hours from shock, but this is rare. Usually he rallies, unless he is already in the last stages of exhaustion consequent on advanced pulmonary phthisis, and his condition may either show little change as the result of the new phase of his malady, or he may gradually sink, or in exceptional cases the occurrence of pneumothorax may tend to retard the progress of the pre-existing tuberculosis. Recovery is possible, especially in cases consequent on empyema (see "Prognosis").

The temperature in pneumothorax shows much variation. In cases where the onset of the condition is attended by much shock, there is usually, as might be expected, a sudden fall followed by a sharp rise. The course of the temperature is most often similar to what is found in tuberculosis.

Physical Signs.—These are usually definite and characteristic.

(*a*) *Inspection.*—The affected side is distended and immobile. The intercostal spaces are flattened. The movements of the sound side are exaggerated. The impulse of the heart may be seen to be displaced towards the sound side. The shoulder on the affected side is raised.

(*b*) *Palpation.*—Vocal fremitus is abolished. The liver and spleen may be felt to be displaced downwards. This is common in pneumothorax, while it is rare in pleural effusion.

(*c*) *Percussion.*—The percussion note is hyperresonant or tympanitic, unless the distension of the side is excessive, when the so-called "tympanitic dulness" may be present. Cardiac dulness is often obliterated.

(*d*) *Auscultation.* — The breath sounds are weak or absent. In some cases they are faintly amphoric. Vocal resonance is diminished.

Certain special auscultatory signs are found in pneumothorax, and are of much assistance in diagnosis. These are: (1) metallic tinkling; (2) the bell sound (*bruit d'airain*); and (3) succussion splash. (1) Metallic tinkling was compared by Laennec to the sound produced when a metallic, glass, or porcelain cup is struck by a pin. He explained it as caused by drops of fluid falling in an air-filled cavity. Its mode of production is doubtful. (2) The bell sound, or *bruit d'airain*, is a highly characteristic chiming sound, produced over a pneumothorax by placing one coin upon the chest wall and striking it with a similar coin. (3) Succussion splash. If the patient is suddenly shaken, while the observer listens through the stethoscope, a splashing sound is often clearly audible.

Metallic tinkling, the bell sound, and the succussion splash simply indicate that fluid is present in a large air-containing cavity, and are not pathognomonic of pneumothorax. They may arise in connection with large phthisical cavities, or a distended stomach, or a distended colon.

The physical signs of pneumothorax may be modified by: (1) the state of the lung on the affected side, *e.g.* by consolidation or excavation; (2) the state of the lung on the sound side (this lung is frequently the seat of disease, and the physical signs will vary accordingly); (3) the amount of fluid present in the affected side.

Diagnosis.—This is usually obvious. Attention to the history and the physical signs rarely leaves much room for uncertainty. Practically, almost the only condition likely to give rise to difficulty is a large, thin-walled pulmonary vomica; but in such cases the heart is not displaced towards the sound side, the side is flattened rather than distended, the bell sound will probably be absent, and the distribution of the physical signs is different from that which prevails in pneumothorax.

Prognosis.—This must have regard both to the antecedent condition and to the special dangers arising from the occurrence of pneumothorax. Two questions present themselves for discussion: (1) What is the prognostic significance of the occurrence of pneumothorax in a case of chronic pulmonary phthisis? and (2) What importance must be attached to pneumothorax supervening upon empyema?

As regards the former question, it may be answered that pneumothorax occurring in the course of pulmonary phthisis usually precipitates the fatal issue. Douglas Powell found the average duration of life in 39 cases of this kind to be only twenty-seven days. In the most favourable case the duration of life was twelve months. Weil found that out of 45 cases about one-half were dead within a month, while some survived for years.

In rare cases the occurrence of pneumothroax seems to retard the progress of pulmonary tuberculosis. The writer has seen one striking case of a young man who decidedly improved after pneumothorax had occurred in the course of chronic phthisis. The patient regained a considerable degree of health, but was subsequently lost sight of.

As regards the second question, pneumothorax supervening upon empyema sometimes—it is difficult to say in what proportion of cases—takes a favourable course. In 2 recent cases in the writer's practice recovery took place. One of the patients was found to be in good health three years subsequent to the occurrence of pneumothorax.

According to Fraentzel, pneumothorax occurring in association with gangrene of the lung is usually fatal within twenty days.

Treatment.—If shock be pronounced at the

outset appropriate measures—such as morphia or ether by hypodermic administration, and the exhibition of alcohol—must be employed. If there be evidence of excessive intrathoracic pressure a fine trocar should be introduced, and some of the contained air allowed to escape. It must not be forgotten that this procedure is attended by the danger that the diminution of the intrathoracic pressure may cause the re-opening of the perforation of the pulmonary cavity in cases where pneumothorax has arisen in advanced phthisis. Hence great caution should be used, and aspiration is not to be employed. If, as is almost invariable, fluid as well as air be present in the pleural cavity, some of it should be withdrawn and examined. Dr. Finlay is of opinion that if the fluid be serous "the general condition of the patient will be no worse than if air alone were present; probably, indeed, better, as the pressure exerted on the lung may tend to check the progress of disease in it, and will promote the effectual sealing up of the perforation. If the liquid be fœtid pus nothing but harm can come from letting it remain in the pleura, and it ought to be evacuated at once." The same authority, referring to cases where the fluid is purulent but not fœtid, advises that, "if the pneumothorax have resulted from rupture of an empyema into the lung, the chest should be freely opened and drained, and the same would hold good if the empyema had ruptured through the chest wall, the opening which nature makes not being, as a rule, sufficient for free drainage. And even in the case of pneumothorax of tuberculous origin a consideration of general principles dictates the free evacuation of the pus." As to the propriety of leaving a serous effusion in pneumothorax undisturbed there can be no second opinion. Such cases are, however, of little practical importance. Fœtid pus should, it is clear, be promptly evacuated by incision and drainage. The treatment of pneumothorax consequent upon empyema is not quite so certain. There is much to be said for the advisability of freely opening the chest in the hope that the pus will thus easily drain from the most dependent part, and that the broncho-pleural fistula will close, but success will sometimes attend an expectant policy. Two of the best cases of recovery which have occurred in the writer's practice were cases belonging to this class, in which for various reasons no operative measures were undertaken. Operation in cases of pneumothorax consequent upon the rupture of a vomica into the pleural cavity is of most doubtful expediency. Many of these cases are already hopeless. The patients, as a rule, are ill fitted to bear operative interference. There is always the bare chance that the occurrence of pneumothorax may not alter the patient's condition for the worse. In view of these considerations, the safer rule would appear to be to recommend operation only when the urgency of the symptoms or the special indications of the individual case would appear to indicate its advisability.

The general treatment of pneumothorax in cases which survive the early dangers of the condition must be on tonic and hygienic lines. In rare cases success may be achieved when little expected.

Malignant Disease of the Pleura.—This subject will be considered in detail in the surgical section. A brief reference to it must suffice in this place. Primary malignant growths are rare in the pleura, but may occur in the form of endothelioma or sarcoma. Secondary deposits are much more frequent. In such cases the seat of the primary affection is usually the lungs, mediastinum, or mamma.

The diagnosis may present considerable difficulties until the case has made progress. The onset is insidious, pyrexia is slight, pain is occasionally present and may be severe, but this is not invariable, shortness of breath and cough are usual, and as time goes on progressive emaciation and debility will probably appear. The physical signs are for a time simply those of effusion. Examination of the fluid may give important indications. It is usually hæmorrhagic, and may contain cancer cells (*see* "Hæmorrhagic Effusion," p. 134). The fluid may, however, be simply serous, and present no distinctive features. At a later stage signs of pressure will probably appear with displacement of organs.

The duration of these cases is from a few weeks to from three to six months.

The treatment must be on general lines. Paracentesis should not be performed unless it is urgently indicated by pressure symptoms. (*See* "Surgical Diseases of the Pleura," *infra*; and "Mediastinum," vol. vi. p. 230 *et seq.*).

Lymphatics of the Pleura.—As in other serous membranes, lymphatic vessels are abundant in and beneath the pleura. They are found in large numbers in the thin layer of areolar tissue underlying the serous covering. "These vessels communicate on the one side by means of stomata with the pleural cavity, and on the other with a network of similar vessels in the interalveolar septa of the lungs" (Quain).

Pleura, Affections of the. B. Surgical

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Injuries of the Pleura.—Severe contusion of the chest, without fracture or external wound, may cause rupture of both pleural layers, and be followed by surgical emphysema (*q.v.*), pneumothorax, hæmothorax, or inflammation of the pleura with effusion. Such an injury will probably be associated with graver damage to the lung, and the symptoms and treatment may, in some measure, be influenced thereby. As the air is mechanically freed from germs in its passage through the lungs it enters the pleural cavity quite pure, and accordingly such injuries, in nearly every case, may be expected to run an aseptic course.

Emphysema of the cellular tissue may be left to nature, as the air becomes absorbed in about a week. It is only when the air is widely diffused and its pressure embarrassing, that it may be dealt with by multiple punctures with a fine trocar.

Pneumothorax thus arising may also be watched, unless the dyspnœa be extreme, when a fine trocar and cannula may be employed for the release of the air. It is not desirable to use an aspirator in either of these conditions.

Hæmothorax, in like manner, should not be interfered with unless the dyspnœa be distressing; and if the aspirator be used for the evacuation of the blood, care should be taken not to draw fresh blood by undue suction. The mechanical assistance of the air and blood in the cavity, with consequent compression and temporary shrinkage of the lung, is favourable to the closure of the rent in the visceral pleura, hence the excuse for non-operative measures, save in extreme dyspnœa.

Lacerations of one or both pleural layers may occur in simple fractures of the ribs or sternum, particularly in those due to direct violence, and the same principles of treatment apply.

Penetrating Wounds.—Wounds of various kinds, including gunshots and compound fractures, admitting air from without to the pleural sac are regarded as penetrating, the parietal layer of the membrane being considered the boundary. Apart from the associated visceral damage, these injuries derive their gravity from septic matters carried in from the surface, scraps of clothing and metallic bodies lodged in the sac. It should be remembered that the pleuræ may be wounded through the abdomen or at the root of the neck.

Traumatopnœa, or breathing through the wound, is a sign of penetration, but it does not necessarily imply wound of the visceral pleura.

In obscure cases it is unwise to probe small wounds lest penetration be thereby caused. It is better practice to enlarge the surface wound and examine it with the finger, if it be desirable to ascertain its depth, or to deal effectively with intercostal hæmorrhage.

Small incised wounds, after thorough antiseptic cleansing, may be closed at once, and if sutures are used they should not include the parietal layer. Larger wounds, and all punctured and gunshot wounds, after similar careful disinfection, should be covered with a copious absorbent antiseptic dressing and binder. It is better not to close them completely, so that drainage may be secured. Many such heal without inflammation. Even gunshot wounds with lodged bullets often do well, as the experience of the South African war abundantly illustrates; and it further teaches that prolonged search by extensive operations for these projectiles is not the best course to adopt. Lodged foreign substances, if readily accessible, should of course be extracted, either through the wound, which may be enlarged for this purpose, or through an independent incision; metallic bodies being located by the X-ray apparatus when available.

In the later management of these cases, should temperature, pulse, and dulness indicate inflammatory effusion, the character of this may be estimated by the exploring syringe. If purulent, arrangements must be made for free drainage, either by enlarging the wound, or, if this be in an unfavourable situation, by an independent opening, according to the principles laid down in the section on empyema.

Tumours of the Pleura.—Primary neoplasms are rare. Secondary growths arising by extension of tumours from the lung, mediastinum, breast, or adjacent bones are not uncommon.

Primary Tumours.	Secondary Tumours.
1. Fibroma.	1. Sarcoma.
2. Sarcoma.	2. Cancer.
3. Endothelial cancer.	

Fibrous tumours have been described by Kingston Fowler and Steven forming broad thickenings of the pleura with dense adhesions to the lung. Their structure is laminated, enclosing small cysts, very dense and glistening on section, and of a pink colour. They are incapable of surgical extirpation.

Sarcomata in all varieties occur, but the round-celled is the commonest.

Endothelial tumours, which are microscopically indistinguishable from true cancers, are supposed to spring from the pleural endothelium, or from the original surface epithelium of the embryo. These either form whitish nodules scattered over the parietal layer, or broad, flat, continuous infiltrations covering the entire membrane.

Diagnosis.—The early symptoms are obscure: shortness of breath, loss of strength and weight, and coughing. There may be slight fever, irregular vomiting, and albuminuria. Pain may be wholly absent, slight, or persistently severe. At a later stage dark or bloody effusion will probably be found, and this may be the first sign to suggest the diagnosis. The fluid may be microscopically examined for cell elements, for it must be borne in mind that

hæmorrhagic effusion may occur in many other states, like tuberculosis, scurvy, nephritis, the exanthemata, and hæmophilia. After aspiration in tumour cases the dulness, diminished fremitus, and resonance may remain. Further, the displacement of the heart or diaphragm is remarkably slight, and there are usually no signs of pressure on the vessels, trachea, or œsophagus. The chest may even be retracted in some cases.

Prognosis is uniformly unfavourable. Most sufferers die in from two to six months, in rare instances they linger for one or two years.

Treatment.—Extirpation is impossible. Even aspiration should not be resorted to very frequently, but only used when the dyspnœa demands.

Exploration of the Pleura

Paracentesis Thoracis—Thoracentesis—Aspiration of the Pleura.—Aspiration or tapping of the pleura may be required in hydrothorax, hæmothorax, chylothorax, pneumothorax, tubercular and malignant effusions, ordinary serous pleurisy, and empyema. It may be regarded as exploratory or curative. The reader is referred to the preceding articles for the indications as to its employment in these conditions.

The operation may be performed with a trocar and cannula, or the cannula may be furnished with a long rubber tube to assist by siphonage. Southey's capillary trocars may be used when a very gradual removal of the fluid seems advisable. All of these methods may fail to empty the pleura, and therefore most operators prefer an aspirator. Two forms of this instrument have their advocates—Dieulafoy's syringe aspirator and Potain's bottle aspirator. The latter is more rapid and less laborious to work owing to the larger vacuum engaged, but Dieulafoy's instrument is the safer, as it can be more accurately gauged and controlled in the event of pain, bleeding, or syncope supervening.

It is assumed that the instruments have been sterilised, and that the hands of the operator and the skin of the patient have been cleansed with soap and water, rubbed with ether or alcohol, and finally moistened with carbolic lotion 1 in 40, or a mercurial lotion 1 in 1000. The patient should be recumbent or nearly so. No general anæsthetic is needed, even in children; and local anæsthesia by freezing or cocaine is also best avoided, as the former hardens the tissues, and the latter may cause a misleading syncope.

The puncture should not be made too low down, lest the abdomen be entered. Any interspace from the fifth to the eighth inclusive on the left side, and either the fifth or sixth on the right, will suit, as the vacuum renders the operation independent of gravitation. The needle should be of good size, $\frac{1}{12}$—$\frac{1}{10}$ inch, and the trocar and cannula mount is preferable to the single needle, as the cannula can be cleared if blocked by coagula. Guarded by the forefinger, and guided by the left forefinger pushed into the interspace, the needle should be confidently thrust through the middle of the space. A hesitating movement is apt to push the thickened pleura before the needle and fail to lodge it in the cavity. For a similar reason a hypodermic syringe, often used in the preliminary exploration, is not to be recommended, as from the shortness and fineness of its needle and feeble suction it may fail to reach and draw a sample of the fluid.

The evacuation should proceed slowly, particularly in left-sided cases in which there is marked displacement of organs; and if bleeding, pain, spasmodic cough, or syncope arise it will be well to suspend or stop the operation. In the event of bleeding it is unwise to resume; but in the three other conditions mentioned, the operator may judge by their severity and persistence whether he may proceed. In the absence of these symptoms, it is best to wholly empty the cavity, as the absorption of the residue, after removal of a part of the fluid, does not occur so frequently as to entitle one to calculate upon its occurrence.

At the close the puncture may be sealed with an antiseptic pad and strapping, or it may be painted with collodion. It is neither necessary nor desirable to bind the chest after aspiration; on the contrary, the respiratory movements should be encouraged.

Difficulties in the operation may arise from close approximation of the ribs, thickening of the pleura, a multiloculated condition, and much coagulation of the fluid. Care should be taken not to wound the heart, pericardium, or the abdominal organs. Wounding of the intercostal vessels need not be feared. Two special risks require more detailed notice, viz. syncope and acute pulmonary œdema. Syncope is not uncommon during the progress of the operation, and is to be promptly combated by diffusible stimulants, nitrites, and by hypodermic injections of strychnia and ether.

Acute œdema of the lung (L'expectoration albumineuse, Terrillon), with copious expectoration of serum, is believed to be due to the sudden release of the lung from pressure, with vaso-motor changes and consequent flooding of the air vesicles with serum strained off from the dilated capillaries. It is an alarming condition and fortunately rare. We are unable "to foresee, prevent, or alleviate it" (S. Paget).

Œdema is more likely to follow the too rapid evacuation of a large effusion, and the onset may be immediate, or be delayed for a few minutes or an hour or two. The duration of the attack may only be a few minutes, or extend to hours or days. The patient becomes cyanosed, oppressed in breathing, with incessant cough, and may expectorate in an hour or two one to

three pints of frothy serum. Fine moist râles are heard over the lung. A fatal termination is not infrequent, but many recover. Prompt treatment by alcohol, the nitrites, and strychnia must be instituted, and dry cupping and oxygen inhalations may also be administered.

Drainage of chronic pleurisies that demand repeated aspiration has been much debated; and although successful cases have been recorded by West, Morison, Wilson, and others it must be admitted that this step has not yet received wide surgical approbation. The almost inevitable conversion of such pleurisies into empyemata, with persistent discharge, renders the propriety of incising and draining these cavities very doubtful. The writer's experience is wholly in favour of depending upon repeated aspiration in such cases.

Empyema.—*Surgical Treatment of.*—It should be remembered that purulent collections in the pleura are usually loculated; in a smaller number of instances the pus occupies the entire cavity; some are multilocular; and a few are bilateral. Of the bilateral effusions one side may contain serous fluid and the other pus.

Recently it has been sought to determine the presence of pus by the X-rays (Walsham, Kilian), but in the present state of our knowledge the aspiration of a sample of the fluid is still needed for proof.

The classification of empyemata into five forms, according to the micro-organism engaged in their production, has an important bearing upon surgical management and prognosis.

1. Pneumonococcus empyema.
2. Streptococcus ,,
3. Saprogenic ,,
4. Tubercular ,,
5. Staphylococcus ,,

The last named is often variously mixed with the other forms, and does not often appear as a separate pathological entity (Netter).

While it cannot be denied that one or more exhaustive aspirations may cure a pneumonococcal empyema, especially in children, it will commonly be found necessary to provide a free opening with drainage, either by incision through an intercostal space or the excision of a segment of a rib. Certainly in streptococcal, saprogenic, and staphylococcal empyemata no delay is warrantable, as the pus is sure to reaccumulate after aspiration, and the risk to life of the retention of these kinds of pus is greater than in the pneumonococcal variety, which is the most hopeful of all. On the other hand, the writer has seen nothing but misfortune result from these open measures applied to tubercular empyema, and he strongly endorses the view that aspiration, repeated only so often as the dyspnœa demands, instead of permanent drainage, is the best course to adopt in these very unpromising cases.

Anæsthesia in these operations is always an anxious question, particularly so in patients with much dyspnœa and displacement of organs, and in those with recently established broncho-pleural fistula. The anæsthesia should never be profound; and indeed, in both its general and local forms it may occasionally be dispensed with. Chloroform or methylene is more suitable than ether, as the latter embarrasses by the coughing and salivation which it induces. A preliminary aspiration to reduce the excessive quantity of pus is often a wise precaution.

The patient should be recumbent, and rolled over on the sound side as little as possible. The opening should be made in the eighth interspace just in front of the scapular angle, or the ninth rib may be resected in the same situation. Efficient drainage both in the erect and lying postures is thus provided, and it will seldom be found necessary to make two thoracic openings as was formerly the practice recommended. In small loculated empyemata the opening must of course be made where the dulness indicates, and in all cases the presence of pus at the selected spot must be verified by an exploratory puncture at the time of operation.

Excision of a rib is superior to mere incision of an interspace, for it supplies an opening which does not become valvular, and which admits a finger for examination of the cavity and lung and the evacuation of large fibrinous coagula. Moreover, the advantage of easy dressing in children and nervous adults is obvious. The risks of the bony section favouring pyæmia, etc., are fanciful.

The Operation.—In incising an interspace the soft parts are to be steadied upon the lower rib, bounding the interspace with the left forefinger and thumb, and an incision two and a half to three inches long is made down upon its upper border, thus avoiding the intercostal vessels and nerve. When all bleeding points have been secured, the pleura is opened by a small incision, which is widened by the insertion of a dressing forceps. The finger is then introduced to prevent a too rapid escape of the pus, and to assist in dislodging coagula.

Excision of the rib involves a three-inch incision boldly carried down to the middle of its outer surface, dividing the periosteum with the other soft tissues. The periosteum is raised towards the rib borders with an elevator for two inches, and the intercostal muscles are detached for a like distance, with due regard to the vessels and nerve, which are practically never seen. The periosteum is then cautiously separated from the deep surface by a lateral movement of the elevator, and a segment of bone, about an inch and a half in length, is clipped out, the elevator being used as a lever to facilitate this step. After securing the bleeding points, the pleura is opened as above described, and the finger is introduced to ascertain the

degree of lung recession and to rake out fibrinous coagula.

At this stage the propriety of washing out the cavity with normal saline solution at 100° Fahr., or boracic acid solution, or other non-poisonous antiseptic, may be considered. The frequent sudden fatalities resulting from such lavage, and the clinical observation that even the most putrid discharges become sweet in a few days by thorough drainage, justify a condemnation of this practice as a routine measure. If ever employed, the irrigating funnel and tube should be used instead of a syringe, and the lotion allowed to flow into the cavity in the gentlest stream, a tube of larger calibre having been inserted to serve as an outflow, in order that no compression of the lung by the irrigating fluid may take place.

No sutures are required for the wound. The permanent drain tube is placed in position and lightly packed around with iodoform or cyanide gauze, a copious absorbent pad of wood-wool tissue is placed over the gauze, and a binder or many-tailed bandage is applied over all.

Drainage tubes of metal, vulcanite, glass, or rubber with a metallic core have been advocated; but a simple rubber tube, one-third or one-half inch in diameter, and furnished with a sheet-rubber or gutta-percha shield to prevent it slipping into the cavity, answers all purposes. Such a tube should have no lateral perforations, save one near the free end, and it should project about one inch into the pleural cavity. There is no advantage in long tubes; but when much flocculent material mingles with the pus, two tubes of slightly different lengths, arranged parallel, may occasionally obviate blocking.

Bilateral empyema should be approached with caution, for, if extensive pleural adhesions have not formed, it will not be safe to open both pleuræ at the same time. One side should be aspirated and the other incised. After an interval of a week or more the aspirated side may be freely opened also.

Treatment after Operation.—The dressings may be changed twice daily for a few days, and later once a day will suffice. The tube, if patent, need not be disturbed for a week. The time at which it can be permanently dispensed with varies with the amount and character of the discharge. One week in children and three weeks in adults may be regarded as minimal. A safer guide is the amount of the discharge; when this declines to half an ounce or less, and becomes almost serous in kind, the tube may be discarded. Godlee's plan of passing a railroad catheter every third day, for a short time thereafter, to prevent accumulation, may be borne in mind. The patient may sit up, if strong enough, in a week or ten days after operation; and may get out of doors in three weeks, provided there is no fever.

Causes of Death in Empyema.—Death may result from increasing embarrassment of breathing in the unrelieved cases. In connection with anæsthesia, operations, or lavage, the catastrophic deaths have already been alluded to. In chronic cases with discharge, septic poisoning, pyæmia with visceral abscesses, hectic fever, amyloid disease, and recurrent hæmorrhages from the giving way of pleural adhesions, may all account for fatal terminations.

Prognosis is very favourable in the acute empyema of children, in whom the pneumococcal form is so common. In adults this form is also very hopeful, if treated early. The streptococcal, staphylococcal, and saprogenic forms are more dangerous; but often do well, if uncomplicated by visceral disease or general septic states. The tubercular variety is practically incurable, in so far as cessation of discharge is concerned, but the fatal issue may be delayed for months or years.

As to the relative frequency of these forms, Netter's analysis of 110 cases showed—32 of the pneumococcal; 51 of the streptococcal, often combined with the staphylococcal; 15 of the saprogenic; and 12 of the tubercular.

Thoracoplasty. — *Estländer's Operation — Thorax Resection.*—Five factors are concerned in the obliteration of the empyema cavity—viz. the expansion of the lung, adhesions between the pleural layers, contraction of the granulation tissue tending to draw the lung and chest wall together, the elevation of the diaphragm, and the falling in of the ribs from atmospheric pressure. To favour the last of these, in intractable cases, Estländer devised the operation of resecting portions of those ribs that bound the cavity. The number and length of the segments requiring removal are determined by flexible probes passed through the sinus, and during the operation by the finger itself.

One long incision over the central rib will admit of three ribs being excised; but, where a larger number of ribs is condemned, several incisions may be needed, or, better, a large flap of the thoracic skin and muscles may be turned up. The ribs are subperiosteally excised for the required distance, and the flap or wound edges sutured, leaving ample drainage openings for tubes or gauze tampons. Schede suggested an actual "thorax resection." Turning up a large musculo-cutaneous flap, with its base upwards, and extending from the fourth rib in front down to the tenth rib in the axillary line, and up along the vertebral border of the scapula, Schede resects the ribs from their tubercles to the costo-chondral junctions. Though the ribs are subperiosteally excised to save hæmorrhage, the periosteum is subsequently excised together with the intercostal muscles and the thickened parietal pleura, forming the whole outer wall of the cavity. He then replaces the scapula and flap, which have mean-

while been strongly retracted. The operation is a severe one, attended by much shock and hæmorrhage, and the results are often disappointing. Schede's tables of his operations, showing 10 cases with 8 cures and 2 deaths, are very encouraging, but the experience in this country, while not unfavourable to its adoption in selected cases, is not such as to lead to so sanguine an estimate of its general applicability and value. It must be conceded that no other operations remedy the spinal curvature or restore the figure in so marked a manner as these extensive resections, but, on the other hand, many of these resections are only partial obliterations of the cavity, the apical portion remaining a suppurating space requiring permanent drainage and resisting all attempts at closure by skin grafting and other plastic measures. The comparative immobility of the two upper ribs is responsible for the failure. The first rib has been resected, but it is considered unwise to tamper with it; and the sinkage of the second on resection is very trifling and the result disappointing. As a guide to the selection of cases for these extensive resections it may be stated, that marked spinal deformity, copious exhaustive discharge, or recurrent hæmorrhage form indications; but the best chance of success is afforded by single cavities of moderate size, bounded by the lower and more movable ribs, in subjects under middle age who are free from tubercular taint.

It must not be forgotten that chronic empyema patients occasionally live for fifteen to twenty years, and may be able to follow their occupations notwithstanding a daily discharge of pus.

Certain difficulties that occasionally complicate the management of empyema remain for consideration.

1. Natural openings in unfavourable situations may have formed, and the advantage of making an artificial drain opening in the situation recommended may in such cases be considered. With our present knowledge, the natural pointing of the empyema ought theoretically never to be seen. Marshall long ago pointed out its frequent occurrence in the second and fifth interspaces in front.

2. Broncho-pleural fistula may exist, and if after a week or two it is evident, from the continuance of septic fever, sweating, and cough, that the cavity retains much pus, a suitable empyema operation has much to recommend it, but caution about administering a general anæsthetic will not be misplaced.

3. Abscess of the brain, "pulmonal cerebral abscess," is said to occur in about 8 per cent of suppurations in the pleura and lung. It is probably pyæmic, and does not lend itself favourably to trephining, for it may be multiple and difficult to localise. If the signs are clear, it would be right to trephine and drain it.

4. A differential diagnosis must be made from pyo-pericardium, subphrenic abscess, and hydatids. Reference to the special articles upon the pericardium and subphrenic abscess may be made for their respective signs, but a brief notice of pleural hydatids is introduced below.

Hydatids in the Pleura may be primary, or they may extend to the pleura from the lungs or liver. The favourite seat is the lower part of the right pleura. They may attain a large size or may burst into a bronchus, the patient coughing up characteristic elements, which, if mingled with bile, point to a hepatic origin. If a hydatid suppurate, the signs and treatment resemble those of empyema, and the prognosis, when similarly incised and drained, is good. Before suppuration signs of a localised collection of fluid having a convex upper outline may be detected, and these signs, associated with considerable pain, but no fever, may excite suspicion of hydatids. Probably aspiration will have been carried out before a diagnosis is made. In some cases, however, transient attacks of slight fever as the hydatid grows may be met with. As regards treatment, it is to be remembered that aspiration in hydatids has special risks of swamping the lung and thus suffocating the patient. If, therefore, a strong probability exist that the disease is hydatids, free incision, with removal of the cyst, should be preferred. In chronic cases wherein the cyst cannot be extirpated prolonged drainage must be employed.

Pleuralgia.—Intercostal neuralgia. *See* Hysteria (*Sensory Disorders*, *Hyperæsthesia*).

Pleurapophysis.—The lateral process of a vertebra or the structure representing it (a rib).

Pleurisy or Pleuritis. *See* Pleura, Diseases of (*Pleurisy*), and *Cross References*.

Pleuro-. — In compound words *pleuro-* (Gr. πλευρά, side, πλευρόν, a rib) means relating to the side, to a rib, or to the pleura; *e.g. pleurocele* (hernia of the pleura), *pleurodynia* (pain in the side), and *pleurospondylian* (having the ribs firmly fixed to the vertebræ).

Pleurodynia.—This term is applied to the pain of a so-called rheumatic nature that affects the muscles and fibro-tendinous structures of the chest wall. It is commonly spoken of as muscular rheumatism. Its ætiology will be discussed later (see "Rheumatism"). The clinical features are fairly distinct, and consist of a sharp, sudden, and often lancinating pain in the intercostal space. In some cases the pain is very acute, and cough is a frequent accompaniment. On physical examination little may be found, but in well-marked cases there is an area of acute tenderness in the intercostal space or in the pectoral or serratus muscles. Pain is aggravated by the respiratory movement. The

differential diagnosis has to be made from intercostal neuralgia and cardiac, pulmonary, or pleural disease. The former is characterised by the presence of localised painful points corresponding to the entrance of sensory nerves; the latter must be determined by looking for the ordinary physical signs of cardiac or pulmonary disease. The local treatment consists in the application of a collodion dressing, or strip of plaster. Some cases are much benefited by counter-irritation. Only in specially severe cases will it be necessary to give an opiate for the relief of the pain. The disease is a constitutional one, and the general treatment will be described under "Rheumatism" (*q.v.*).

Pleuromelus.—A teratological type in which an accessory limb springs from the side of the thorax.

Pleuroperitoneal.—Affecting or belonging to both the pleura and the peritoneum. *See* Tuberculosis (*Morbid Anatomy*).

Pleuro-Pneumonia.—Inflammation affecting both the pleura and the lungs, especially a contagious disease in horned cattle, with this character. *See* Food (*Characteristics of Unsoundness*); Milk (*Pathological*).

Pleuroprosoposchisis.—Lateral fissure of the face; a genal cleft due to imperfect closure of the fissures between the fronto-nasal, superior maxillary, and mandibular processes. *See* Cheek, Fissure of.

Pleurosomatoschisis.—A teratological type in which there is gastroschisis (open condition of the trunk) in the side; congenital lateral aperture of the trunk.

Pleurosomus.—A teratological type characterised by lateral fissure of the chest wall with or without prolapse of the lungs and heart through it.

Pleurospasm.—Cramp in the side.

Pleurothotonos.—The arching of the body to one side due to spasm of the muscles of that side.

Pleurotomy.—Incision into the pleura.

Pleurotyphoid.—Typhoid fever complicated by pleurisy. *See* Typhoid Fever (*Symptoms, Pleuro-typhoid*).

Plexiform.—Resembling a plexus, *e.g. plexiform neuroma*, a disease in which numerous tumours affect the nerves.

Pleximeter.—A disc or plate of ivory (or other substance) placed on the body in mediate percussion; it is struck by the *plexor* or hammer.

Plexus.—In Anatomy a plexus is a structure made up of interwoven nerves, vessels, or fibres, *e.g.* the *brachial* plexus, the *cœliac* plexus, the *hypogastric* plexus, etc.

Plica.—A fold, *e.g.* the *plicæ vaginæ* (vaginal rugæ), or the *plica semilunaris* (a fold of conjunctiva of the eye).

Plica Polonica s. Saxonica.—A morbid state of the hair of the scalp, of the beard, or of some other part of the body, regarded at one time as a special disease but now known to be due to want of cleanliness; the hair is filthy and is matted together into a stiff, tangled mass, in which parasites abound; it was common at one time in Poland, Posen, and Galicia. *See* Pediculosis (*Pediculosis Capitis*).

Plimmer's Bodies.—Intracellular bodies found in cancerous tissues, and once regarded as the causal agent of cancer.

Plombières. *See* Balneology (*France*); Mineral Waters (*Thermal*).

Plugging. *See* Abortion (*Treatment of Inevitable Abortion, Plugging the Cervix*); Labour, Post-Partum Hæmorrhage (*Treatment, Curative, Plugging the Uterus*); Labour, Obstetric Operations (*Induction of Premature Labour*); Pregnancy, Hæmorrhage (*Unavoidable Hæmorrhage, Treatment, Champetier Bag*); Puerperium, Pathology (*Sudden Death, Plugging of Pulmonary Artery*); Uterus, Inflammations of (*Chronic Metritis, Treatment*).

Plugs.—Vaginal plugs or tampons are applications consisting of cotton-wool, iodoform gauze, marine lint, etc., introduced into the vagina with the view of checking hæmorrhage, supporting the uterus, or relieving inflammation; they often contain some medicinal substance such as glycerine, ichthyol, or a styptic.

Plumbism.—Lead-poisoning. *See* Toxicology (*Lead*); Trades, Dangerous (*Lead-Poisoning*); Water (*Diseases produced by Impure Water*).

Plumbum. *See* Lead and *Cross References* (vol. v. p. 377).

Plummer's Pill.—A pill containing calomel, sulphuretted antimony, guaiacum resin, castor oil, and alcohol; pilula hydrargyri subchloridi composita of the B.P.

Plural Pregnancy.—Twin pregnancy or a gestation in which two or more fœtuses are present in the uterus; a *pluripara* is a woman who has given birth to twins or (more frequently) one who has had more than one pregnancy. *See* Labour in Multiple Pregnancy; Pregnancy, Multiple.

Plutomania.—A form of insanity or

monomania in which the patient believes himself to be extraordinarily wealthy.

Pneobiognosis.—The test whether a child has been born alive or dead, by the presence or absence of air in the lungs. *See* Medicine, Forensic (*Infanticide, Hydrostatic Test*).

Pneograph.—An instrument for graphically indicating the characters of the respiratory current of air (Gr. πνέω, I blow).

Pneumarthrosis.—Air or gas in a joint.

Pneumato-.—In compound words *pneumato-* (Gr. πνεῦμα, air) means relating to air, to gas, or to respiration; *e.g. pneumatometer*, an instrument for measuring the amount of air taken into and expelled from the lungs during inspiration and expiration.

Pneumatocele.—A tumour or hernia containing air or gas, *e.g.* a pneumatocele of the neck. *See* Neck, Region of (*Cysts and Cystic Tumours*).

Pneumatosis.—The presence of air or gas in organs or tissues of the body from which it is usually absent or in which it is normally present in small amount, *e.g.* in the stomach in nervous dyspepsia. *See* Indigestion (*Nervous Dyspepsia*).

Pneumatotherapy.—The treatment of disease by compressed air.

Pneumaturia.—The presence of free gas in the urine. *See* Urine, Pathological Changes in (*Pneumaturia*).

Pneumo-.—In compound words *pneumo-* (Gr. πνεῦμα, wind or air) means relating to the lungs or to respiration; *e.g. pneumolith* (a concretion in the lung), *pneumorrhagia* (pulmonary hæmorrhage or apoplexy), and *pneumotyphus* (typhus fever accompanied by pneumonia).

Pneumobacillus.—The bacillus of Friedländer, which may be found along with or apart from the pneumococcus in cases of pneumonia. *See* Pneumonia, Bacteriology (*Other Bacteria*).

Pneumococcus.—*See* Pneumonia, Bacteriology. *See also* Bone, Diseases of (*Osteomyelitis due to Pneumococcus*); Conjunctiva, Diseases of (*Acute Ophthalmia, Etiology*); Ear, Acute Inflammation of the Middle (*Causes, Micro-organisms*); Expectoration (*Parasites, Pneumococcus*); Fluids, Examination of Pathological (*Bacteriology*); Meninges of the Cerebrum (*Acute Simple Meningitis, Micro-organisms*); Meningitis, Epidemic Cerebro-Spinal (*Bacteriology*); Nose, Examination of the (*Secretion, Bacteriology*); Peritoneum (*Pathology, Bacteriology*); Peritoneum, Tuberculous Peritonitis (*Pneumococci*); Pleura, Diseases of (*Bacteriology of Pleural Effusions*); Post-mortem Methods (*Bacteriological Investigations*); Septicæmia (*Definition, Bacteria*); Stomach and Duodenum, Diseases of (*Infective Gastritis, Bacteria*); Teeth (*Bacteriology of the Mouth*); Tonsils, Diseases of (*Tonsillitis, Causes*).

Pneumoconiosis. *See* Lungs, Pneumonokoniosis.

Pneumoderma.—Emphysema of the subcutaneous tissues.

Pneumogastric.—Literally the term means relating to the lungs and to the stomach, but most commonly it is the vagus nerve which is intended when the word pneumogastric is employed. *See* Vagus Nerve. *See also* Epilepsy (*Symptoms, Epigastric Aura*); Heart, Physiology of (*Cardiac Nervous System*); Larynx, Neuroses of (*Innervation of the Larynx*); Pharynx, Examination of (*Neuroses of Sensation*); Physiology (*Cranial Nerves, Circulation, Respiration*, etc.); Stomach and Duodenum, Diseases of (*Anatomy, Nerves*).

Pneumograph.—An instrument for recording graphically the movements of the chest in respiration.

Pneumohæmothorax.—The presence of air and blood in the pleural cavity.

Pneumohydrometra.—The presence of air or gas and serous fluid in the uterus.

Pneumohydropericardium.—The presence of gas and fluid in the pericardial sac.

Pneumohydrothorax.—The presence of gas and fluid in the pleural cavity.

Pneumomassage.—A form of treatment of chronic otitis media by inflation of the tympanic cavity with air and by simultaneously producing a rarefaction of the air in the external auditory meatus.

Pneumonia.

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Bacteriological Section

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See also Adrenal Glands, Adrenalin (*Use in Pneumonia*); Alcohol (*Clinical Uses, Indications*); Alcoholism (*Complications*); Anæsthesia, General Physiology (*Ether - Pneumonia*); Anæsthesia, Ether (*Broncho - Pneumonia*); Arteries, Ligature (*Common Carotid, Complications following Ligature*); Asphyxia (*Causes, Catarrhal Pneumonia*); Brain Affections of Blood-Vessels (*Thrombosis, Causes*); Bronchi, Bronchitis (*Acute and Chronic, Diagnosis*); Colon, Diseases of (*Membranous Colitis, Secondary, Causes*); Cough (*Clinical Varieties*); Delirium Tremens (*Prognosis, Pneumonia*); Expectoration (*Sputum in Various Diseases, Pneumonia*); Hæmatemesis (*Diagnosis*); Hæmoglobinuria (*In Fevers*); Hæmoptysis; Hiccough (*Etiology*); Hydropathy (*Acute Diseases, Pneumonia*); Immunity (*Serum in Pneumonia*); Influenza (*Effects, Pneumonia*); Insanity, Nature and Symptoms (*Etiological Varieties, Pneumonia*); Joints, Diseases of (*Pyogenic Diseases in Pneumonia*); Leucocytosis (*In Pneumonia*); Liver, Tropical Abscess (*Diagnosis*); Lung, Tuberculosis of (*Precedent Illnesses, Pneumonia*); Lungs, Pulmonary Fibrosis (*Chronic Interstitial Pneumonia*); Lungs, Gangrene of (*Etiology*); Lungs, Syphilis of (*Congenital Pneumonia*); Mediastinum (*Growths, Diagnosis*); Meninges of the Cerebrum (*Acute Meningitis, Diagnosis*); Meningitis, Epidemic Cerebro-spinal (*Complications*); Mumps (*Complications*); Nephritis (*Etiology, Clinical Features*); Nerves, Multiple Peripheral Neuritis (*Etiology*); Nose, Accessory Sinuses, Inflammation of (*Etiology*); Oxygen (*Uses*); Peritoneum, Acute Peritonitis (*Diagnosis*); Plague (*Symptoms*); Pregnancy, Affections and Complications (*Pulmonary Disturbances, Pneumonia*); Pulse (*In Pneumonia*); Purpura (*Symptomatic, Infectious*); Rheumatism, Acute (*Symptoms, Pneumonia*); Rheumatism in Children (*Symptoms, Pneumonia*); Suppuration (*Etiology, Micro-organisms*); Syphilis (*Visceral, Lungs*); Therapeutics, Serum Therapy (*Antipneumococcic Serum*); Tuberculosis (*Broncho-pneumonic Tuberculosis*); Typhoid Fever (*Symptoms and Complications*); Typhus Fever (*Symptoms and Diagnosis*); Urine, Pathological Changes in (*Acetonuria in Pneumonia*).

Introductory.—The term pneumonia is properly applied to any inflammatory process affecting the lung tissue, just as pleurisy is employed to designate any inflammation of the pleural membranes, or as hepatitis and nephritis are applied to any inflammation of the liver and kidneys respectively. Moreover, as there may be acute or chronic parenchymatous or interstitial forms of hepatitis and of nephritis, so there may be corresponding forms of pulmonary inflammation or pneumonia. In the more important forms of chronic pneumonia the interstitial changes especially predominate, while in the acute pneumonias the more characteristic lesions are as a rule due to the implication of the pulmonary parenchyma. Any acute inflammation of the lung, therefore, is an acute pneumonia, but it is usual to recognise several varieties.

Sometimes the classification is based on anatomical differences in the resulting lesions. When the inflammatory process is specially concentrated around the smaller bronchi, and follows their lobular distribution, the term broncho-pneumonia or lobular pneumonia is employed. The term catarrhal pneumonia is often applied to the same process, owing to the fact that the exudation into the pulmonary alveoli largely consists of catarrhal cells. More rarely the acute inflammatory changes select the connective tissue framework of the lung, giving rise to an acute interstitial form of pneumonia, such as is met in some forms of influenzal pneumonia and in pleuro-pneumonia of cattle. A third type is recognised under the name of acute lobar, or fibrinous, or croupous pneumonia, when the inflammatory process involves large continuous tracts of lung tissue, such as an entire lobe, or the greater part of a lobe, and when the exudation into the pulmonary alveoli is at first and for the most part composed of fibrin. As a matter of fact, however, there are commonly met forms of acute pneumonia which do not conform to any one of these anatomical types, but present very variable and mixed lesions. Even among those which are rightly classified as lobar forms of acute pneumonia there may appear very striking differences as regards the precise anatomical lesion.

Other forms of acute pneumonia are differentiated with special reference to the causal agent associated with them. Such specific forms as the tubercular inflammations of the lungs, or such forms as the suppurative pneumonia of pyæmia, present, as a rule, definite types with characteristic anatomical lesions. But when we try to apply this ætiological differentia to other forms of pneumonia we encounter the difficulty that the same cause may give rise to anatomically different pulmonary lesions. Thus a group of acute pneumonias due to the pneumococcus would include both broncho-pneumonic and lobar types. A similar difficulty is presented by the forms of pneumonia that appear in influenza, plague, and many other conditions of microbic infection. It is, therefore, better, in place of attempting an exact definition or a precise outline of the scope of this article, merely to state that in discussing the pathology of

acute pneumonia it is understood that the term is limited to certain forms of acute pulmonary inflammation, all microbic in origin, and probably all due to or associated with Fraenkel's pneumococcus, but that all forms of the typical lobar type are necessarily included.

Historical.—Long before any bacterium had been associated with acute lobar pneumonia as the causal agent it had been suspected that this condition was infective and analogous in every way to the acute specific fevers. Its occurrence in epidemic form, and some instances apparently of direct infection, together with the definite course of the fever, all tended to support this view. But it was not until 1883 that the first important contribution to the bacteriology of acute pneumonia was made.

In that year Friedländer published a paper which attracted much attention, and in which he claimed to have obtained in pure cultures the causal microbe of pneumonia. This, he described as a coccus, usually occurring in pairs, and surrounded by a definite capsule. They were found in the lung tissue, bronchi, etc., in cases of acute pneumonia. They could be isolated and grown on peptone gelatine at the ordinary room temperature. From this fact alone it is obvious that the coccus described and isolated by Friedländer is not the same as that which is now accepted as the most important ætiological factor in acute pneumonia and is associated with the name of Fraenkel. In fact the organism which Friedländer at first described as a coccus has now come to be regarded as a short bacillus, and is commonly known as Friedländer's pneumobacillus. Inoculation of animals with Friedländer's organism, however, in many instances caused death from septicæmia with inflammation of the serous membranes, thus demonstrating the pathogenic capacity of the organism in certain conditions. One of the most important results of Friedländer's work was the stimulus it afforded to the study of the bacteriology of pneumonia.

In the same year (1883) Talamon described a coccus which he obtained from the sputum and pulmonary exudation in acute pneumonia, and which from its shape he termed "lanceolatus." This organism he was able to cultivate in bouillon, but usually in an impure condition, and inoculation of rabbits produced a septicæmia often associated with a fibrinous pericarditis and pleurisy. There can be little doubt that this organism is the same as the pneumococcus later described and isolated by Fraenkel.

As early as 1880 certain observations were made in America by Sternberg and in France by Pasteur on the presence in the saliva in health and in disease of micrococci, and on the fact that inoculation of rabbits with saliva gave rise to a "sputum septicæmia," in which the blood teemed with micrococci. The significance of these observations was not at first appreciated.

After a preliminary communication in 1884 Fraenkel published in 1886 a very full and accurate description of the characters of the pneumococcus—noted its lanceolate outline, its occurrence in pairs, and its capsulated appearance. He succeeded in isolating pure cultures, and found that both in its cultural and pathogenic characters the microbe differed from that previously described by Friedländer. In particular, Fraenkel's organism did not grow on gelatine at the ordinary room temperature, and it was highly pathogenic to rabbits, in both these respects contrasting with Friedländer's organism. Another important advance made by Fraenkel was the identification of the pneumococcus with the "coccus of sputum septicæmia" previously described in healthy saliva by Pasteur and Sternberg.

In the same year (1886) Weichselbaum independently published the results of a still more extensive research on the bacteriology of acute pneumonia, including 94 cases of the typical lobar form and 35 other cases of acute pulmonary inflammation. From these he isolated four organisms: (1) The "diplococcus pneumoniæ," which he described as an oval or lanceolate coccus, corresponding in morphological, cultural, and pathogenic characters with Fraenkel's pneumococcus; (2) the "streptococcus pneumoniæ," which, on the whole, presented similar characters, but was rounder and formed longer chains; (3) the "bacillus pneumoniæ," which he regarded as identical with Friedländer's pneumobacillus; (4) the "staphylococcus pyogenes aureus." Of these organisms the "diplococcus pneumoniæ" was present in the great majority of cases, the "streptococcus" was much less common, and the "bacillus pneumoniæ" rarer still. He was led to the conclusion that while the great majority of cases of acute pneumonia are due to the "diplococcus pneumoniæ" (Fraenkel's pneumococcus), some are caused mainly by a streptococcus, and a few by the "bacillus pneumoniæ" (Friedländer's pneumobacillus). How far the streptococcus pneumoniæ of Weichselbaum corresponds with the ordinary streptococcus pyogenes, and how far with Fraenkel's pneumococcus, is left somewhat uncertain; and what is of more importance, the reader is left uncertain as to the data on which his conclusions are based, in particular, whether they have reference to acute lobar pneumonia in its limited acceptation or acute pneumonia in its widest sense.

Since these earlier researches were published the attention of many observers has been devoted to the elucidation of problems involved in the relation of micro-organisms, especially the pneumococcus, to acute pneumonia. Among the later contributors may be noted Gamaleia, who investigated in particular the pathogenic

properties of the pneumococcus in experimental inoculation; Netter, who examined a great number of cases with the object of determining the distribution of the pneumococcus and its relation to various morbid states; Eyre and Washbourn, who perfected the culture media, and the latter of whom was successful in establishing a condition of artificial immunity.

The Pneumococcus (Fraenkel).—It is obvious from these researches that whether this organism is admitted to be the essential causal agent in the pathogenesis of acute pneumonia, or whether it acts thus only in the majority of cases, it is of all the microbes concerned by far the most important. A brief outline of some of its morphological, biological, and pathogenic characters is a necessary preliminary to any discussion regarding its rôle in the production of disease in man, and the whole question of its relationship to disease introduces one of the most interesting problems which bacteriology has ever attempted to solve.

In form the pneumococcus appears as minute oval or lanceolate bodies, usually in pairs and often in chains, the free extremities of each pair being pointed. In animal tissues and fluids each pair of cocci appears surrounded by a capsule, due to a peculiar swelling of the cell envelope, and this capsule may be stained by special methods. In any circumstances, but especially in artificial culture, the cocci show a marked tendency to degenerate and undergo involution; but even when this degeneration has advanced to such an extent that the bodies of the cocci have largely or entirely disappeared, the capsules often persist and may be identified by suitable methods of staining. The significance of this lies in the fact that at such a stage cultures would probably remain sterile, and inoculations would probably have negative results, so that the recognition of the capsules might be the only means of determining the presence of pneumococci. In contrast with the pneumobacillus of Friedländer it should be noted that the pneumococcus retains the stain in Gram's method.

In cultivation the organism is somewhat fastidious, and practically requires to be incubated at or about the body temperature. On blood serum, blood agar, or ordinary agar it occurs as minute translucent colonies which tend to remain separate. On all ordinary media cultures rapidly degenerate, rapidly die, and subcultures soon lose their virulence. Eyre and Washbourn, however, by the most careful attention to certain conditions of cultivation, were enabled to maintain both the vitality and the virulence unimpaired for several months.

Both within and without the animal body the pneumococcus is quite exceptional in its brief duration of life and in the rapidity with which its virulence is lost. In artificial culture this is very striking, and presents a considerable difficulty in the way of experimental work. Sometimes the virulence may be intensified or restored by passage through a series of rabbits. In susceptible animals the organism may live longer in a virulent condition than in cultures, but even then it is apt to die sooner than most pathogenic microbes. In old empyemas, in the later stages of pneumonia, etc., a large number of devitalised and degenerated pneumococci are often found, but occasional instances are met where the virulence has been maintained for months.

Fraenkel and Weichselbaum had stated, and their views have been confirmed by subsequent observers, that pneumococci suffer a progressive diminution in virulence as the course of a pneumonia advances. Netter, indeed, goes so far as to say that in the day of the crisis the virulence of the sputum is often suddenly lost, and remains in abeyance for two or three weeks. Apropos of this question Welch of Baltimore says that attenuation of virulence within the human body probably does occur, inasmuch as pneumococci derived from inflammatory exudates may be devoid of any pathogenic effect in animals. From the late stages of pneumonia which had gone on to recovery, and at the time of the crisis, he had obtained pneumococci which were as virulent and sometimes more virulent than those procured from earlier stages of the same case. He points out that in experiments of this nature the number and character of living pneumococci inoculated must be considered. It is known that the pulmonary exudate contains many dead cocci, and that the living cocci vary greatly among themselves in degree of virulence. Moreover, when animals are inoculated with material obtained directly from the human body, there may be introduced along with the pneumococci toxic or immunising substances which influence the course of the injection. It may indeed be shown that such material, though proved to contain very virulent pneumococci, may not produce any pathogenic effect.

Pathogenic Effects of Experimental Inoculation.—The results of experimental inoculation of animals with the pneumococcus show extreme degrees of variation. The actual number of cocci injected, the presence or absence of other bacteria or toxins, and the site of inoculation, very materially condition the result. But the two factors on which this variability chiefly depends are (1) the virulence of the microbe and (2) the susceptibility of the animal.

(1) Both in natural conditions and in artificial cultures pneumococci may have lost their virulence to such an extent as to be quite innocuous. On the other hand, by such methods as passage through a series of susceptible animals, the virulence may be so intensified that a few cocci injected into a rabbit will produce a fatal septicæmia in less than twenty-four hours. Between

these extremes pneumococci of all degrees of virulence may be met, so that the possibilities of variation in the lcisons produced by this cause alone are obviously considerable. Of course if the resistance of the animal is increased the effects are similar to those due to attenuation of the virus. Thus a rabbit which has received a subcutancous inoculation of virulent pneumococci usually dies in about forty-eight hours of an acute septicæmia. In such cases the local inflammatory reaction at the site of inoculation is always slight, but the blood is found to be swarming with enormous numbers of capsulated pneumococci. If, however, a rabbit which has been partially immunised receives a similar injection, or if a somewhat less virulent organism is employed, the animal may live for a week before it succumbs to the infection. Here the local reaction is more intense, so that at the site of inoculation the tissues show an extensive area of inflammatory œdema, hæmorrhage, and frequently also necrosis. The fluids at the local site are crowded with pneumococci, but the blood may contain few or none, or may show the results of a septicæmia which has immediately preceded death. A third and entirely differcnt result may be obtained if the rabbit be still more highly immunised, or if the pneumococci be still more attenuated. In such conditions a local focus of suppuration forms at the site of inoculation, and the animal usually recovers. Hence in the same species of animal, the rabbit, by suitable modifications of its powers of resistance or of the virulence of the pneumococci, subcutaneous inoculations may result in such divergent lesions as (*a*) an acute septicæmia with a negligible local reaction, (*b*) an intense local cellulitis, œdema, and necrosis with little tendency to septicæmia, and (*c*) a localised abscess of varying size.

(2) When the susceptibilities of different orders of animals are also considered, the interpretation of the pathogenic effects of the pneumococcus becomes still more complicated. Among the most susceptible are mice and rabbits, and these animals when inoculated beneath the skin, in the peritoncal sac, or in the lung, with virulent pneumococci, almost invariably die of an acute septicæmia with little or no local reaction. Less susceptible are guinea-pigs, sheep, and dogs, in which animals subcutaneous inoculation results in an extensive local cellulitis and œdema which does not invariably lead to a fatal result. If, however, in a partially immunised rabbit, or in the more immune animals, such as the sheep or the dog, the injection of pneumococci be not subcutaneous, but intrapulmonary, then again an intense local inflammation ensues at the site of inoculation, which in this case is the lung, and an acute fibrinous pneumonia and pleurisy result. "When a dose of pneumococci sufficient to kill a rabbit is injected subcutaneously in the human subject, it gives rise to a local inflammatory swelling with redness and slight rise of temperature, all of which pass off in a few days. It is therefore justifiable to suppose that man occupies an intermediate place in the scale of susceptibility, probably between the dog and the sheep, and that when the pneumococcus gains an entrance to his lungs the local reaction in the form of pneumonia occurs" (Muir and Ritchie).

Intoxication and Immunity.—It is a noteworthy fact that few cases of pneumonia succumb to asphyxia, so that the extent of lung involved is not in most instances the direct cause of death. What is probably of more effect in determining a fatal issue is the degree of nervous prostration, and especially of cardiac failure. After death, in addition to the pulmonary lesion there is invariably evidence in the condition of the heart, spleen, kidneys, and other viscera, that the patient has suffered from the effects of an acute general intoxication. During life, moreover, many of the more urgent symptoms are referable to such a condition. It rarely happens that any general invasion of the blood by pneumococci occurs except just before death. It is therefore more than probable that pneumococci which invade the lung and there produce an acute local inflammation, also elaborate some diffusible toxic substance which, absorbed into the blood-stream, gives rise to the general toxæmia of an acute pneumonia. The nature of these toxic bodies is not definitely known, but, without going into details, one may say that all experimental work on the subject supports this view of their existence and highly poisonous nature.

Artificial immunisation of susceptible animals has also been effected by various observers, including G. and F. Klemperer and Washbourn. Moreover, it was found that the blood serum of animals artificially immunised was itself protective, in other words, that a true antitoxin was produced. It is supposed that the crisis so typical of the recovery from an acute lobar pneumonia occurs only when the antitoxin has been developed in such abundance as completely to neutralise the toxin. In this relation two facts are of significance as supportiug the view that the pneumococcus is thc essential cause of acute lobar pneumonia. (1) The serum of patients who have recovered from such an attack is to some extent protective against the results of artificial inoculation with the pneumococcus. (2) The serum of an animal artificially immunised by injection of cultures of pneumococci has a beneficial action in cases of acute lobar pneumonia.

Distribution of the Pneumococcus in Health and in Disease.—Reference has already been made to the fact that the saliva of healthy persons injected into a rabbit may produce a fatal septicæmia, due to the presence of pneumococci. The existence of virulent pneumococci

in the mouth in health has obviously a most important bearing in the pathogenesis of acute pneumonia.

In the production of diseased conditions it is not easy to exaggerate the rôle of the pneumococcus, for the multiplicity and frequency of the lesions that may be ascribed to its action are indeed remarkable. Pulmonary inflammations are undoubtedly the most common effect, and, in the opinion of the writer, pneumococci may be found in every case of acute lobar pneumonia, in most cases of broncho-pneumonia, and in most of those more or less mixed forms of pneumonia which are complicated by the associated action of other organisms, such as influenza, plague, typhoid, diphtheria, etc. In cases of typical lobar pneumonia they are practically the only organisms present in the affected lung, and are found in greatest abundance in the fibrinous exudation into the pulmonary alveoli and in the pleural surface. They are most readily detected in and isolated from the more recent parts of the pulmonary lesion, as in the older parts they are usually much degenerated. In the purulent softening and other suppurative changes that appear in the lung in many fatal cases of pneumonia, they may be the only organisms present, or they may be associated with other pyogenic bacteria, especially streptococci.

Next to the lungs the cerebro-spinal membranes are most frequently attacked, and this may occur not only as a sequel to or a concomitant of pneumonia, but as an apparently independent condition. The resulting meningitis is sero-purulent and usually very widespread, involving both cerebral and spinal membranes. The serous cavities of the thorax and abdomen are frequently the seat of pneumococcal infection by direct extension from an affected lung, but they too may show an apparently primary and independent inflammation. Empyema of pneumococcal origin is comparatively common, and, in the experience of the writer, most cases of fibrinous pericarditis with sero-purulent effusion are due to pneumococci, whether an antecedent pneumonia can be traced or not. Primary peritonitis of pneumococcal origin is uncommon, but a recent peritonitis in fatal cases of pneumonia is by no means rare. In children one of the most common effects is an otitis media, and in children, too, broncho-pneumonic lesions due to pneumococci are relatively more frequent than in adults.

In the more virulent forms of pneumonia the lesions produced by the pneumococcus tend to become multiple, and the condition more nearly approaches the septicæmic type of infection, as seen most typically in mice and rabbits. In such cases not only may lungs and pleuræ be affected along with the pericardium and the mediastinal tissues, but more remote lesions are frequently associated, *e.g.* meningitis, endocarditis, arthritis, etc., and in some instances pneumococci are found in considerable numbers in the circulating blood.

Summary and Conclusions. — One result of these laborious researches is to show that the pneumococcus produces in man a local lesion, usually pulmonary, with general toxic effects. Sometimes the local lesion takes the form of a meningitis, an otitis, an empyema, or a pericarditis as an apparently independent condition. This is really the same disease as acute lobar pneumonia, with an accidental difference in the site of inoculation and consequent local inflammatory reaction, and many of the symptoms are similar from similar toxic effects. Hence it not infrequently happens that a pneumococcal pericarditis or empyema is mistaken for an acute pneumonia. Occasionally, however, the pneumococcus acquires increased virulence, and produces a condition more allied to the septicæmia of mice and rabbits.

It is difficult to avoid the conclusion that the pneumococcus is the essential cause of acute lobar pneumonia, as well as of the many other inflammatory conditions with which it is associated. But the versatility of this remarkable organism renders the proof much more complex than in the case of pathogenic bacteria. At first sight, too, Koch's elementary postulates for establishing a causal relationship appear to be all evaded by the pneumococcus. For it cannot be said to be present in all cases of acute pneumonia, and in a few instances even of typical lobar forms it has been sought in vain. Moreover, it is present in the mouth and upper respiratory tract in health as well as in disease. Again, although it has been isolated in pure cultures, inoculation of animals does not reproduce the identical lesions found in man. And, finally, there is no consistent lesion even in the human subject, and not even in the lung.

The allegation that the pneumococcus is occasionally absent even in typical cases of acute lobar pneumonia is one which is open to doubt. For, if the statement is founded on a microscopic examination, it might be that none of the cocci would be visible, having undergone degeneration until only capsules were left. Again, if based on the negative results of cultures, the fallacy might be due to the devitalised condition of the cocci. Even if reliance is placed on the negative results of experimental inoculation of lung tissue, it must be remembered that, after a time, the virulence of pneumococci in the animal body is often quite lost, or that antagonistic substances, of the nature of antitoxins, may have been injected along with the cocci, and so neutralised their effect.

The fact that pneumococci are usually present in the saliva of healthy persons has proved a stumbling-block to many writers, but this is precisely the fact required to explain the in-

cidence of pneumonia from exposure to cold, and as a sequel to debilitating diseases and exhausting fevers. Apparently the cocci in the buccal and nasal secretions are not sufficiently virulent to produce any pathogenic effects in health. But, when the resistance is lowered by some depressing influence, the organisms are enabled to infect the tissues and develop inflammatory conditions. From their anatomical positions the lungs would be most easily infected, but the readiness with which an otitis or a meningitis might be developed from the nares is also obvious.

The contention that inoculation of pure cultures does not sufficiently reproduce the disease in animals is quite untenable when the following points are considered:—(1) The effects of inoculating pneumococci of varying virulence in animals of varying susceptibility; (2) the fact that, in the human subject, the lesions admittedly due to pneumococci are almost as varied as those produced in animals by artificial means; (3) the analogy with such organisms as the bacillus anthracis, which causes in the susceptible ox or guinea-pig an acute septicæmia, and in the more immune human subject an intense local reaction.

The fact that even in the lung in man there is no constant lesion—the pneumococcus being associated sometimes with an acute lobar pneumonia, sometimes with a broncho-pneumonia—is more difficult to explain. It has been suggested that variations in virulence and in susceptibility might modify the result, and probably also the precise mode of entrance to the lung would condition the anatomical form of the lesion.

Other Bacteria associated with Acute Pneumonia. — Although, as has been shown, there is good reason to believe that the pneumococcus is the essential cause of all typical forms of acute lobar pneumonia, there are many other conditions in which acute pneumonia may arise. Some of these forms are broncho-pneumonic in type, others are of a mixed character, and of both of these it may be said, either (*a*) that they are due to the pneumococcus alone—broncho-pneumonic forms of pneumococcal infection especially predominating in children, or (*b*) that they are due to other bacteria associated with the pneumococcus, or (*c*) that they are due to other bacteria alone. Among the various bacteria which may be the cause of acute pneumonia (other than typical lobar forms), and which may act either in conjunction with or independently of the pneumococcus, are Friedländer's pneumobacillus and those associated with the following diseases: influenza, plague, diphtheria, enteric fever, septicæmia, and pyæmia.

Friedländer's pneumobacillus is of special interest as being one of the original claimants for supremacy in the production of acute lobar pneumonia. In 1886, when the fundamental papers of Fraenkel and of Weichselbaum appeared, the former concluded that the pneumococcus was the sole cause of true lobar pneumonia, while the latter held that about 5 per cent was due to the pneumobacillus alone. Each view still has its own supporters, but most recent work is in the direction of showing that the pneumobacillus, though frequently associated with the pneumococcus, is not the essential factor in typical lobar forms. A short bacillus, usually arranged in pairs, and in body fluids and tissues usually encapsulated, it differs from the pneumococcus in not retaining the stain by Gram's method, and in growing more readily in culture, forming the "nail" growth in gelatine. The fact that it more readily adapts itself to a saprophytic existence offers a probable explanation of the cases in which this organism alone has been cultivated from acute lobar pneumonia, for at the time when the lung tissue or secretion is examined, the pneumococcus may be so devitalised as not to grow in artificial culture, and not to infect animals on inoculation. It must be admitted, however, that the pneumobacillus is distinctly pathogenic to certain animals; rabbits appear to be immune, but guinea-pigs and mice may die of an acute septicæmia, so that the pneumobacillus may play an important part in human disease, especial acute pulmonary inflammations, though not the essential cause of lobar pneumonia.

In the acute pneumonia consecutive to influenza, the type of the lesion is mainly broncho-pneumonic, with acute interstitial changes. In the lungs and sputum the influenza bacillus has been found alone, but more frequently it is found associated with the pneumococcus. In the intense hæmorrhagic forms of broncho-pneumonia that may occur in plague, it often happens that plague bacilli alone are found in the lung and sputum, but in many cases pneumococci too are present. In the broncho-pneumonia secondary to diphtheria, pneumococci are frequently present, associated with either diphtheria bacilli or streptococci, or both. In the course of enteric fever a mixed form of pneumonia may occur, in which typhoid bacilli may be present alone or along with pneumococci. In septic and suppurative pneumonias pyogenic bacteria are sometimes the only varieties found, but more frequently there is superadded an infection by the pneumococcus.

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Definition.—By the term "pneumonia" is to be understood, for the purposes of this article, a specific general infection of bacterial origin, analogous to the other infective or so-called "zymotic" diseases. This infection either begins in the air vesicles of the lungs, a primary growth of the infecting micro-organism taking place in the pulmonary parenchyma as in a congenial soil, or else it shows a special affinity for that anatomical structure, in which it and its toxins find a secondary focus and become localised, so leading to topical, inflammatory, and sometimes organic degenerative changes. The local inflammation is signalised by increased blood-supply (or fluxion), followed in turn by hyperæmia of the affected lung substance; an exudation into the air vesicles, which become choked with it; secondary degenerative changes in that exudate; and, finally, its absorption and expulsion.

The clinical phenomena attending such an infection are, first, general; secondly, local. The general symptoms are those of a continued fever setting in after an incubation period of variable though short duration—chills, headache, rheumatoid and neuralgic pains, loss of appetite, constipation, elevation of temperature, prostration. The local subjective signs, or symptoms, are pain in the chest, hurried, difficult breathing, cough, and finally expectoration of a peculiar kind. The local objective signs, or physical signs, are such as to suggest the plugging of greater or smaller tracts of pulmonary tissue with inflammatory products, the signs changing with each progressive stage in the disease, as will be hereafter described. Evidences are usually forthcoming that neighbouring parts—the pleura on the one hand, the bronchial mucous membrane on the other—share in the mischief wrought by the disease.

Pneumonia, or pneumonic fever, as it should be more rightly called, must be regarded as a multiple infection. That is to say, it arises from one or other of many infections, as will be afterwards shown. To the classical type of acute croupous pneumonia, or lobar pneumonia, however, two micro-organisms especially seem to stand in a direct causal relation. These are —(1) The *Diplococcus pneumoniæ* of Fraenkel, so called by Weichselbaum, now called the "pneumococcus"; and (2) the *pneumococcus* of Friedländer, or the *Bacillus pneumoniæ* of Flügge, by which latter name it is now usually known.

As to the communicability of pneumonia no doubt can any longer be entertained. The spray blown from the mouth with each cough and the dried expectoration are the most probable, as they are the most obvious, channels of infection.

Usually affecting a lobe or lobes of either lung, acute pneumonia in children and in the aged is often disseminated, assuming a lobular rather than a lobar character in such patients. Like typhus fever, pneumonia is a cyclic and self-limited disease, in which the constitutional fever runs its course in five, six, or seven days, when (in uncomplicated cases) it terminates by a crisis.

Historical.—Pneumonia, taken in the widest acceptation of the term, may be described as a malady of all the centuries, of all climates, and of all ages of life. Its epidemic prevalence, according to August Hirsch, is recorded in the sixteenth-century chronicles of pestilence in Italy, Germany, the Netherlands, Switzerland, and France. One of the earliest allusions to epidemic malignant pneumonia is met with in Savonarola's *Practica*, published at Venice in 1497, where it is stated: "In civitate nostrâ Paduæ et Trevisii et Venetiæ 1440 de mense Martii antea et post apparuit *pleuresis mala*, quæ erat contagiosa, ex quâ multi et plurimi moriebantur."

In the second book of his treatise, "Περὶ νούσων," Hippocrates gives a faithful account of pneumonia under the name "Ἡ Περιπλευμονίη." The later form of the word was "περιπνευμονία," used by Celsus and Galen. Celsus (Lib. iv. cap. iii.) says: "Vehemens et acutus morbus quem περιπνευμονικόν Græci vocant." Plutarch uses the shortened form πνευμονία, the Attic being properly πλευμονία, Hippocrates using the equivalent ἡ πλευμονίς.

Sydenham in the seventeenth century described true pneumonia under the name *peripneumonia vera* as contrasted with bastard peripneumony—*peripneumonia notha*—which Huxham in 1764 defined as an obstruction of the lungs by a heavy, viscid, pituitous matter. Was this œdema of the lungs, or (as Dr. Pye Smith states in his article on "Pneumonia" in Dr. Clifford Allbutt's *System of Medicine*) bronchitis?

To Hoffmann belongs the credit of introducing the name *pneumonic fever*, which modern pathology and symptomatology alike justify and approve. To the German school, represented especially by Cohnheim, we owe the term *fibrinous* or *croupous pneumonia*, which means that a coagulable fibrinous exudation takes place on the free surface of the affected lung, that is, into the air-vessels of the affected portion of the organ. *Pleuro-pneumonia* is a valuable name for the disease, in so far as it emphasises the frequency with which the pleura shares in the morbid process. Lastly, the adjectives

"acute," "sthenic," "asthenic," "typhoid," or "ataxic," "massive," and so on, find a place in the terminology of pneumonia, pneumonitis, or lung fever.

In the valleys of the High Alps in Switzerland a severe form of pneumonia in spring is known as "Stich," or "Alpenstich" (*i.e. stitch*).

The infectiousness of pneumonia was observed by Dunns in 1592. He wrote: "Contagiosum quoque esse inde liquido constabat, quod multi in eadem familiâ corriperentur." Ravicio, writing at Venice in 1613, uses the words "influsso epidemico maligno con qualche immediato contagio," of an outbreak "osservato nella terra di *Guastalla.*" The term *hepatisation*, as applied to one stage of the pathological anatomy of pneumonia, was apparently suggested by Morgagni, who in 1761 used the words—"Pulmones compactam *ut in hepate est*, substantiam habebant." Pirri found, in an epidemic at Rome in 1779, "I polmoni degenerate in una sostanza epatica per la sua durezza."

According to Hirsch, the earliest records of malignant epidemical inflammation of the lungs in Spain, England, Denmark, and North America belong to the eighteenth century. In France and Switzerland many epidemics of pneumonia occurred during that century, the season of nearly all the outbreaks being winter and spring, and the type of the disease being "typhoid" or "ataxic," sometimes "bilious."

The literature of pneumonia grew apace as decade followed decade in the nineteenth century, which also witnessed the elucidation of the etiology of this *genus* of disease, for pneumonia includes many species. Huxham long ago said: "There are very different degrees, I might say species, of this disease, which demand a particular attention and a method of cure peculiarly adapted to each."

Epidemics of pneumonia occur from time to time. Huxham described an outbreak of a severe type which visited Plymouth in the latter part of 1745 and the beginning of 1746. A pandemic of the disease overran North America between 1812 and 1825. Laennec described an "Epidemic Pneumonia" as occurring among the conscripts of 1814. An outbreak commenced in the Akerhus prison, Christiania, on 18th December 1866, lasting until May 1867. Among an average of 360 prisoners in that period, 62 cases occurred, of which 15 ended in death. Dr. L. Dahl and Professor W. Boeck attributed the outbreak chiefly to overcrowding. To the same cause Dr. Bryson ascribed a very remarkable epidemic of pleuro-pneumonia which broke out in some ships in the Mediterranean fleet in 1860. Dr. Thoresen of Eidsvold, Norway, met with a local outbreak of "croupous pneumonia" in a single row of cottages near some glassworks in the summer of 1869, when the town was otherwise almost free from the disease. In the spring of 1874 an epidemic of "sewer-gas pneumonia," as it was aptly called, prevailed at East Sheen, Mortlake, London, S.W., in connection with the opening of a ventilator in a main sewer. The cases occurred in a first-class boys' school facing the road under which the sewer ran. During the summer of the same year pneumonia, apparently traceable to sewer-gas poisoning, prevailed in Dublin. It was described under the name of "pythogenic pneumonia" in a paper written conjointly by the late Dr. Thomas Wrigley Grimshaw, C.B., afterwards Registrar-General for Ireland, and by the present writer. In the autumn, winter, and spring of 1874-1875 an epidemic of pneumonia swept over the south-western counties of England (Wynter Blyth). A serious outbreak at Middlesbrough-on-Tees in 1888 formed the subject of an admirable report by Dr. Edward Ballard, Inspector of the Local Government Board, England. The outbreak lasted for twenty-four weeks, comprised about 1000 cases, and caused 369 deaths. In April and May 1890 there was an outbreak at Scotter, in Lincolnshire, in which 22 deaths occurred among 32 known cases, a mortality of 69 per cent. This epidemic was admirably described by Dr. T. B. Franklin Eminson, medical officer of the Scotter district of the Gainsborough Union, and it was also specially investigated by the Local Government Board inspector, Dr. Parsons, who attributed the outbreak to sewer-air emanations and simple contagion conjointly. In the early spring of 1893 epidemic pneumonia attacked the township of Yeadon, Yorkshire, with a population of 7500. Dr. C. J. Russell M'Lean, D.P.H., medical officer of health for the township, observed 35 undoubted cases, of which 24, or 71·4 per cent, occurred in the months of March and April. There were 13 deaths, the case mortality being as high as 37·14 per cent. Dr. M'Lean cites two series of cases—3 in each instance—in which the disease was apparently communicated from person to person.

A startling fact as to the frequency and mortality of pneumonia is brought out by Dr. Hector Mackenzie, of St. Thomas's Hospital, who finds that in a population of 30,000,000 in England and Wales, at least 220,830 persons are annually affected with pneumonia, and that 31,950 persons annually die from this disease, the mortality being thus 14·5 per cent of those attacked. In the year 1898 the death-rate from pneumonia in England and Wales was 1323 per million males in the population, and 947 per million females. Tables 20 and 22 in the *Sixty-first Annual Report of the Registrar-General of Births, Deaths, and Marriages in England* (1898) show that the annual death-rate per million from pneumonia among males, which had been as low as 1089 in the year 1881, rose with a bound from 1206 in 1889 to 1731 in 1890, and still further to 1798 in

1891, coincidently with the first epidemic or epidemics of influenza. Similarly among females the annual death-rate, which had been down to 740 in 1881, rose from 848 in 1889 to 1095 in 1890, and to 1165 in 1891. These figures show what a far-reaching effect influenzal pneumonia has upon the death-rate from "pneumonia" in the abstract.

In 1898, out of a total of 82,404 deaths registered in Ireland, 3550 were referred to pneumonia—2074 in males, 1476 in females. That is to say, pneumonia was responsible for 4·2 per cent of all the deaths, and the death-rate due to it was equal to 1 in 1279·9, or 0·78 per thousand of the estimated population.

Etiology.—Pneumonia, taking the term in its full acceptation, must be looked upon as an essential or specific febrile disease, strictly analogous to diphtheria, enteric fever, smallpox, or any other of the recognised infective diseases (*Infectionskrankheiten*). It is a microbic infection in which the lungs are the usual, though not the invariable, battle-ground. In 1899 J. Pelnar reported two cases of pneumococcus sepsis without pneumonia. The first was in an infant three months old, presenting fever and convulsions. The child died, and at the autopsy gastro-enteritis, purulent meningitis of the convexity, and purulent rhinitis were found, cultures from the meningeal pus and the spleen giving the Fraenkel-Weichselbaum diplococcus. The second case was that of a woman, aged 22 years, in the puerperium. There was an uncertain diagnosis of scarlet fever with phlegmonous tonsillitis, followed by sepsis and leptomeningitis, cultures from the spleen and from the tonsillar and meningeal pus yielding the pneumococcus. Pneumonia was absent in each case.

The varying phenomena presented in different cases of pneumonia probably depend, as it has been suggested, on a varying virulence of its supposed specific micro-organisms, the affection being regarded as an essential disease analogous to diphtheria, enteric fever, smallpox, or any other infective malady. The micro-organisms which appear to stand in a direct causal relation to pneumonic fever have already been named. But there is evidence to show that the *Diplococcus pneumoniæ* and the *Bacillus pneumoniæ* are not exclusively concerned in producing inflammation of the lung tissues.

The subject may be considered from both an etiological—or, we might say, a bacteriological—and a clinical standpoint. Naturally, it is chiefly from the latter point of view that hospital physicians have had opportunities of regarding the question. It is impossible, however, to ignore the bacteriological aspects of the case.

In support of Flügge's statement just quoted, we propose to adduce evidence that the micro-organisms peculiar to erysipelas, to influenza, to tuberculosis, and to enteric fever may one and all give rise to a specific pneumonia or pneumonic fever. So also may Löffler's diphtheria bacillus (the *Bacillus pestis* of Kitasato and Yersin) and the bacillus of malignant anthrax, as well as other pathogenic bacteria. Flügge, for example, says: "We are already acquainted with pneumonias which are caused by aspergillus and actinomyces; it is *a priori* not improbable that also among bacteria there are several other species which can set up pneumonia."

I. Erysipelas.—In the form of this disease which has been called "erratic" or "vagrant erysipelas" (*erysipelas migrans*)—the *erysipèle ambulant* of French writers—the attack may be protracted for one or two months. In such cases not only every part of the surface of the body, but the whole tract of mucous membranes, and even the lungs and pleuræ, may in turn become affected. Dr. Petèr, of Paris, has drawn attention to the spread of erysipelatous inflammation from the pharynx to the respiratory passages, causing in sequence bronchitis, bronchiolitis (capillary bronchitis), and pneumonia.

In a case observed by the writer at Cork Street Fever Hospital, Dublin, many years ago, the converse of this happened. A man was admitted suffering from *pneumonia migrans*. After some days a blush of erysipelas showed over one shoulder, and spread thence down the back, with the interesting result that simultaneously the pneumonic symptoms subsided. So great was the impression made upon him by this case that ever since the writer has recognised the propriety of looking upon *erysipelas pulmonum* as a distinct species of the great genus Pneumonia.

Grisolle quotes from Serres a case of a patient who had several attacks of pneumonia, each terminating in an attack of erysipelas. Wilson Fox states that he had seen only one such case. The erysipelas appeared three days after complete defervescence, and the resolution of the pneumonia and the subsequent recovery of the patient were greatly protracted.

The teaching of Levy, of Strassburg, that *Streptococcus pyogenes* is an exciter at once of suppuration and of erysipelas, is now generally accepted. This pyogenic bacterium was obtained by Fehleisen from the skin involved in cases of erysipelas in 1883, and by Rosenbach and Passet from the pus of acute abscesses within a year or two afterwards. Sternberg gives the following synonyms for *Streptococcus pyogenes*—micrococcus of erysipelas (Fehleisen), *Streptococcus erysipelatis*; streptococcus of pus, *Streptococcus longus* (von Lingelsheim).

If, then, we admit the identity of the pus-producing streptococcus with that of erysipelas, we at once obtain a key to the occurrence of an acute pneumonia in erysipelas. For this very bacterium—the *Streptococcus pyogenes*—plays a

part that is second to none in the production of influenza-pneumonia.

Prof. Hermann Lenhartz, of Hamburg, in his masterly monograph on Erysipelas in Hofrath Prof. Hermann Nothnagel's *Specielle Pathologie und Therapie* (vol. iii. part 3), considers that pneumonia is to be regarded as erysipelatous only when it has arisen in immediate connection with a cutaneous, pharyngeal, or laryngeal erysipelas. Mosny, however, saw a pneumonia appear in the servant of a gentleman ill of facial erysipelas. The attack terminated fatally within two days. At the autopsy a circumscribed broncho-pneumonic area was found, from which pure cultures of streptococci were obtained. These produced a true erysipelas in the ear of a rabbit. Denuce and Schönfeld found the specific streptococci of erysipelas both in the pulmonary capillaries and in the pericardial and pleural exudations in cases of pneumonia occurring in the course of erysipelas.

Waldenburg and Friedreich have emphasised the connection between the so-called "wandering pneumonias" and erysipelas. A serpiginous advance of the pulmonary hepatisation, a marked tendency to involvement of both lungs, the uniform occurrence of marked splenic enlargement, and a frequent coincidence of acute erysipelas and "puerperal fever" caused Friedreich to speak of these cases as "erysipelatous pneumonias."

Lenhartz, in quoting the foregoing authors, while not denying the possibility of primary erysipelatous pneumonias, considers that they are extremely rare. It is, however, certain that such pneumonias may be caused by the *Streptococcus erysipelatis*, and not by the *Diplococcus pneumoniæ* of Fraenkel, when they accompany or follow an external erysipelas. It may, in his opinion, remain doubtful whether a direct transmission of the germ from the skin to the lungs has caused the pneumonia, or whether the infection has occurred through the blood. In the latter case the erysipelatous pneumonia is to be simply interpreted as a septicæmic manifestation.

II. Influenza.—In the article on this disease (vol. iv. p. 429) it was pointed out that the pneumonia of influenza, while producing the ordinary physical signs of acute croupous pneumonia, is often latent in its course or accompanied by a profuse muco-purulent expectoration, with scarcely any rusty sputa. Leyden describes the pneumonias observed by him in influenza as running a peculiar course: severe pain in the side and dyspnœa were rarely noticed, the typical sputum was often wanting, the local process was often atypical—being of a migratory, catarrhal, and of a lobular distribution in many instances. The foremost German authorities on the influenza of 1889-1890 laid stress on the erysipelas-like spread of influenza-pneumonia in the lungs, and on the prominent part which the *Streptococcus pyogenes* seemed to play in its etiology. The question, however, in such cases is probably one of a secondary infection, for which the attack of influenza merely laid the foundation. Certainly the discovery by Pfeiffer, in 1892, of the *Bacillus influenzæ* in the purulent bronchial secretion, and by Canon in the blood of patients suffering from epidemic influenza, must be regarded as conclusive proof of the existence of a specific primary infection to which all other infections are accidental or secondary.

III. Tuberculosis.—We do not here allude to acute tubercular fever (in which the lungs may escape unscathed) on the one hand, or on the other to the local peripneumonic processes which accompany sporadic depositions of tubercle in the lungs in ordinary catarrhal phthisis. Nor do we refer to those cases in which in the wake of an acute primary croupous pneumonia the wounded lung falls a ready prey to a secondary infection by the *Bacillus tuberculosis*, when this micro-organism finds a fertile soil in the caseating exudation of an unresolved pneumonia. These several conditions are all beside the present question.

Our concern is with acute phthisis, or scrofulous pneumonia, and the so-called acute tuberculo-pneumonic phthisis. Both these affections present consolidations in the lungs of a pneumonic origin, but tuberculisation, as well as pneumonia, exists.

In both these varieties of "Consumption" we have examples of true pneumonia resulting from an infection by the *Bacillus tuberculosis* of Koch.

IV. Enteric Fever.—It is well known that pneumonia is more commonly observed as a complication in enteric fever than in typhus. Murchison noted it in 13 out of 100 cases, and Austin Flint (according to Bartlett) in 12 out of 73 cases. It commonly occurs in the third or fourth week, but may usher in the disease. In this latter case its presence is probably an indication that the enteric fever poison has entered the system through the lungs. It is most commonly a *lobular pneumonia*, but occasionally it occurs under the form of ordinary *croupous pneumonia*.

It is, indeed, true that Eberth points out that anatomical investigations had (up to 1881) afforded no evidence of the admission of the *Bacillus typhosus* through the lungs. With this Gaffky does not agree, for he considers it highly probable — or at least the possibility cannot be contested—that the lungs may occasionally represent the seat of invasion. Eberth himself quotes a case observed by W. Meyer, of Berlin, in which death ensued on the second day of illness. In this case there were found at the autopsy hyperæmia of the lungs, spleen, and kidneys, in the lower portion of the ileum marked swelling of the solitary follicles and

Peyer's patches. Microscopical examination revealed a very exceptionally large deposit of Eberth's bacilli in the cells of the submucosa and in the intermediate muscular layers of the intestine. Apparently they were not found in the lungs, notwithstanding their hyperæmic condition.

Even if we take it as not yet proved that infection in enteric fever may occur by way of the lungs, there is no doubt that a close correlation exists between this disease and that variety of acute pneumonia, or pneumonic fever, to which the term "Pythogenic Pneumonia" has been commonly applied since 1875, when the late Dr. Thomas W. Grimshaw, afterwards Registrar-General for Ireland, and the present writer read a paper on the subject before the Medical Society of the College of Physicians of Ireland.

In the eighth volume of the *Johns Hopkins Hospital Reports* (Baltimore, 1900) will be found an analysis and general summary of the cases of enteric fever—829 in number—treated in the medical wards of the Johns Hopkins Hospital from 1889 to 1899. It is from the pen of Professor Osler. Speaking of complications affecting the respiratory system, Dr. Osler says :—

"One may divide pneumonia in typhoid fever into two groups, according to its appearance with the onset, during the course, or towards the termination of the disease." He proceeds : "*Pneumonia at Onset.*—The interesting feature of this form, the so-called pneumo-typhoid of the French, is that the cases may present all the characteristics of ordinary croupous pneumonia, no other diagnosis may be reached, and it may not be corrected until autopsy." He recalls the fact that he had in 1894 given the history of a remarkable case of this character, in which clinically he had no suspicion that there was any other disease than pneumonia, but the autopsy showed characteristic enteric fever. He reports a very similar case, occurring in a man aged 68, who died within the first forty-eight hours without any suspicion having been entertained that he had enteric fever. The autopsy showed the lesions of this disease. Again, in the case of a woman aged 26, who was admitted on November 11, 1898, complaining of cough, headache, and pain in the back, the symptoms remained chiefly pulmonary for nearly eleven days, and Dr. Osler and his colleagues did not recognise the case as one of enteric fever. There was no leucocytosis at first. On November 13 there were 7600 white corpuscles; on November 22, 23, and 24 they increased to 22,000; on November 28 there were 12,500. The patient had no diazo reaction throughout. The Widal test was applied repeatedly, but it was not until November 24 that it could be said to be fairly positive. Then on the 26th a crop of rose spots and the general features clinched the diagnosis.

As regards enteric fever, the influence of season and weather in determining pneumonic trouble is, no doubt, considerable; but it cannot be accepted as paramount or exclusive. And, indeed, it is reasonable to suppose that in some cases, at all events, the pneumonitis is directly due to a localisation of the specific poison of enteric fever in the lung, whether that poison be the *Bacillus typhosus* itself, or a toxin derived therefrom.

V. Anthrax.—"That man may be infected with anthrax by way of the respiratory organs," writes Sternberg, "seems to be well established. In England the disease known as 'wool-sorters' disease' results from infection in this way among workmen engaged in sorting wool, which is liable to contain the spores of the anthrax bacillus when obtained from the skin of an animal which has fallen a victim to this disease. That infection occurs through the lungs is shown by the fact that these organs are first involved, the disease being, in fact, a pulmonic anthrax."

That, in infective diseases in general, infection may occur through the mucous membranes of the respiratory tract, has been demonstrated (according to Sternberg) by several bacteriologists—especially by Buchner, who caused mice and guinea-pigs to breathe an atmosphere containing in suspension a powder consisting of dried anthrax spores mixed with lycopodium powder or pulverised charcoal. In a series of 66 experiments, 50 animals died of anthrax, 9 of pneumonia, and 7 survived. Microscopical examination of sections and culture experiments showed that the lungs were extensively invaded.

VI. Epidemic Cerebro-Spinal Meningitis.—The *Diplococcus intracellularis meningitidis* of Weichselbaum occasionally produces a specific pneumonia. In a report on an epidemic of cerebro-spinal meningitis which prevailed in Boston, Massachusetts, and the neighbouring towns, from the spring of 1896 to that of 1898, published by the Massachusetts State Board of Health, Councilman, Mallory, and Wright mention 8 cases in which the diplococcus caused pneumonia, in addition to which there were 2 cases of croupous pneumonia with pneumococci, 7 of broncho-pneumonia, and 13 cases of congestion with œdema.

VII. Plague.—The most fatal form of this dread disease is that in which it declares itself as a true pneumonia, albeit more frequently of a disseminated kind (broncho-pneumonia), of a lobular rather than of a lobar type. It is not merely deadly, but dangerous, because of the vast crowds of bacilli which are found in the expectoration, whereby the infection may be widely diffused.

During the epidemic of plague in Bombay in 1897, Captain L. F. Childe, I.M.S.,[1] observed

[1] Report to the Plague Research Committee in Bombay.

that, concomitantly with the increase of mortality from the bubonic form of the disease, deaths from pneumonia multiplied *pari passu*. In the pneumonic form of plague, according to Childe, the only marked evidences of disease are found in the lungs; the lymphatic glands and other organs are scarcely affected at all. In these cases there was general engorgement, with considerable œdema and a reddened condition of the bronchial mucous membrane. A number of pneumonic patches, varying in size from a pea to an egg, were found scattered through the lungs. These patches had the appearance of the first and second stages of lobular pneumonia, but no patches were found which had passed on to the third stage of softening and breaking down. Petechial hæmorrhages were usually forthcoming on the surface of the lung. The bronchial glands were either enlarged, swollen, œdematous, soft, and distinctly engorged, or else they were small and of the usual appearance—perhaps a little engorged.

In cases of plague pneumonia Childe states that the bacilli of plague were seen in abundance in the pneumonic areas. They could be found in profusion amongst the catarrhal epithelial cells and leucocytes which filled the alveoli and terminal bronchioles, as well as among the blood corpuscles of the alveoli into which hæmorrhage had occurred. Similarly in the lungs of non-pneumonic cases they could be seen, but in far fewer numbers, and mostly where small hæmorrhages had taken place into the alveoli.

Dr. James Cantlie says that pneumonic plague is usually ushered in by a rigor, dyspnœa, cough, a high temperature, and marked prostration. The sputum is profuse, of a watery character at first; it may be tinged with blood from the onset, but more usually blood does not appear until the illness has lasted twenty-four hours. The pulse is soft and compressible from the first, and cardiac distress is considerable. Pneumonic patches—lobular pneumonia—occur in either lung and at any portion of the lungs, but are more usually met with in the lower lobe and towards the posterior aspect. Moist sounds, especially at the base, and bronchial rhonchi are to be heard, but all the signs and symptoms are peculiarly evanescent, and a patch of pneumonic consolidation, dull to-day, clears up to-morrow, when another part of the lung becomes affected. Delirium, frequently of a violent type, is present; but before death coma supervenes, preceded, it may be, by a muttering delirium. The pneumonic variety of plague is a most rapidly fatal form of the disease, the patient usually dying on the third or fourth day.

In an article on "Pneumonia and Broncho-Pneumonia," published in February 1900, the editor of *The Practitioner* (Mr. Malcolm Morris) wrote as follows: — "Certain pathologists, without contesting the intervention of the pneumococcus in the majority of cases of lobar pneumonia, consider that this microbe is not always *en cause*; that other micro-organisms can give rise to this disease. To unity in cause, they oppose plurality (Weichselbaum, Jürgensen, Finkler). Their opinion is founded on this consideration, that in a pneumonic focus other pathogenic microbes than the pneumococcus have been found; that these microbes introduced into the lung of animals have given rise to inflammatory lesions."

In contradistinction to these views, which we venture to adopt from our own clinical experiences, Netter says dogmatically: "There is no pneumonia without pneumococci." He also states that "the constant presence of the pneumococcus of Talamon-Fraenkel in lobar pneumonia, and the plurality of microbes causing broncho-pneumonia, are the distinctive elements which bacteriology permits us to recognise in the history of pulmonary inflammations." According to Netter, the pneumococcus of Talamon and Fraenkel is the specific microbe of *lobar* pneumonia, but he will surely admit that the pneumococcus plays a leading part in the causation of pneumonia in children, yet how seldom is its distribution lobar. Nor is the lung by any means the only seat of a primary localisation of the pneumococcus. In 121 post-mortem examinations Netter himself found the primary localisation to lobar pneumonia in 65·95 per cent, broncho-pneumonia and bronchiolitis in 15·85, meningitis in 13, purulent pleurisy in 8·53, otitis media in 2·44, endocarditis in 1·22, and abscess of the liver in 1·22 per cent.

Predisposing Causes

1. *Town life* increases both the prevalence of and the mortality from pneumonia. The Registrar-General for Ireland (Mr. Robert Matheson, LL.D., M.A.) has kindly furnished tables showing the number of deaths from pneumonia by ages and sexes registered in the whole of Ireland and in the Dublin Registration District respectively during each of the five years 1894-1898, together with the average deaths from pneumonia for the same period. From these tables it appears that the average annual death-rate per 10,000 of the population from pneumonia throughout Ireland in the quinquennium 1894-1898 was 7·8. The corresponding figure for the Dublin Registration District was 14·4. So that practically the pneumonic death-toll was twice as heavy in the Irish metropolis as it was throughout the country at large. In other words, pneumonic affections kill twice as many of the population in a large town compared with the open country.

2. *Age.*—From the same tables we learn that the average annual deaths from pneumonia in all Ireland during the five years 1894-1898 were

3574. Among these were 395 deaths of infants under 1 year of age, 245 of children aged 1 year, 103 of children aged 2 years, 65 of children aged 3 years, and 39 of children aged 4 years —in all 847 deaths of children under 5 years of age, or 23·7 per cent. Between the ages of 5 and 10 years the deaths were 111; from 10 to 15 years, 71; from 15 to 20, 104; from 20 to 25, 124; from 25 to 35, 259; from 35 to 45, 335; from 45 to 55, 437; from 55 to 65, 571; from 65 to 75, 463; from 75 to 85, 215; from 85 to 95, 35; and at 95 years of age and upwards, 2.

3. *Sex* plays an important part in the etiology of pneumonia. In England and Wales, in 1898, 1323 males out of every million died of pneumonia, while the corresponding number of females was only 947. Three-fifths of the deaths from this disease registered in Ireland in the five years 1894-1898 inclusive were among males, two-fifths among females. The exact numbers were—males, 2111 deaths, or 59 per cent; females, 1463 deaths, or 41 per cent; total deaths, 3574. Sex, however, is rather an accidental than a positive factor in the etiology, for it is only after the age of 20 years that the disproportion in the deaths among the sexes becomes extreme. Under 1 year old, indeed, out of 395 deaths, only 166 were of females, whereas 229 were of males, the percentages being—females, 42; males, 58. Between the ages of 1 and 2 years the respective deaths were —males, 135; females, 110; total, 245; or, in percentages, males, 55; females, 45. Then comes a change—between 2 and 3 years the sexes die of pneumonia in equal numbers—males, 52; females, 51; total annual deaths, 103. Between 3 and 4 years the numbers are —males, 30; females, 35; total, 65; between 4 and 5—males, 18; females, 21; total, 39. The above figures give 847 deaths yearly, of which 464, or 55 per cent, occur among males, 383, or 45 per cent, among females. But between 5 and 10 years, 57 females die of pneumonia for every 54 males; and between 10 and 15 years, 39 females for every 32 males. Taking these ten years—5 to 15—together, we find that of 182 yearly deaths only 86 occur among males, but 96 among females, the percentages being—males, 47; females, 53. After puberty, *i.e.* 15 years, the sexes draw more and more widely apart as regards the deadly influence upon them of pneumonia, the balance being altogether against the male sex. The explanation is obvious. After the critical age of 15 males are more exposed to the other predisposing causes of pneumonia, including over-exertion, over-heating, vicissitudes of atmospheric temperature, wet and cold, and, above all, alcoholic intemperance and injury.

4. *Season.* — Contrary to what one might *a priori* imagine, pneumonia is not so much a disease of winter as of spring, and of late spring especially.

August Hirsch, Professor of Medicine in the University of Berlin, after pointing out that pneumonia, even in its narrowest acceptation of fibrinous or so-called croupous pneumonia, is an anatomical term that includes several inflammatory processes differing from one another in their etiology, goes on to observe that the prevalence of the malady depends very decidedly upon certain influences of season and weather. He gives an elaborate table of percentages of pneumonic prevalence in the several months at a large number of places in Europe and America. According to this table, the largest number of cases falls in the months from February to May, the smallest number in the period from July to September. Taking the average for all the places mentioned in the table, it appears that 34·7 per centum of the patients were attacked in spring (March to May inclusive); 29·0 in winter (December to February); 18·3 in autumn (September to November); and 18·0 in summer (June to August). The combined percentage for winter and spring is 63·7; that for summer and autumn is 36·3. If the number of cases in summer be taken as 1, then autumn has 1·02, winter 1·6, and spring 1·9. Nearly all the recorded epidemics of pneumonia have occurred in winter and spring. From the foregoing considerations, Hirsch confidently concludes that the origin of the malady is dependent on weather influences proper to winter and spring, and more particularly on *sudden changes of temperature and considerable fluctuations in the proportion of moisture in the air.* He holds that any exceptionally large number of cases of "inflammation of the lungs" at the other seasons, more especially in summer, has coincided with the prevalence of the same meteorological conditions phenomenally at that season.

Netter thinks that the maximal prevalence of pneumonia in March, April, and May is due to an increased virulence of the pneumococcus during those months.

In a paper on "Pythogenic Pneumonia," published in 1875 by the late Dr. Grimshaw and the writer, will be found a table, compiled from the returns of the Registrar-General for Ireland, which shows the number of deaths from bronchitis and pneumonia registered in the Dublin Registration District in each quarter of the nine years 1865-1873 inclusive. According to that table, of every 100 deaths from bronchitis, 44 on the average occurred in the first quarter of the year, 22 in the second, only 10 in the third, and 24 in the fourth quarter. Thus the mortality from bronchitis was twice as great in the first as it was in the second quarter, and more than four times greater in the first than in the third quarter.

Very different were the facts as to pneumonia —of every 100 deaths from this disease, 32 on the average occurred in the first quarter, 27 in the second, 16 in the third, and 25 in the fourth

quarter. The mortality from pneumonia was only *one-fifth* greater in the first than in the second quarter, and only twice as great in the first as in the third quarter. The extreme winter fatality of bronchitis and its low summer fatality were equally wanting in the case of pneumonia.

A careful analysis of the weekly returns of the Registrars-General of England and Ireland for ten years ending with 1885, and of the same returns for the year 1886, brings out a similar remarkable contrast between bronchitis and pneumonia, as to the time of year when these diseases are respectively most prevalent and fatal in London and Dublin.

In both cities bronchitis falls to a very low ebb in the third or summer quarter of the year (July to September inclusive), when only 12 per centum of the deaths annually caused by this disease take place in Dublin, and only 11 per centum in London. In the last or fourth quarter (October to December inclusive), the percentage of deaths from bronchitis rises to 27 in Dublin and to 30 in London. The maximal mortality occurs in the first quarter (January to March inclusive), when it is 38 per centum in both London and Dublin. In the second or spring quarter (April to June inclusive), the deaths from bronchitis declined to 23 per centum in Dublin and to 21 per centum in London.

The mortality from "pneumonic fever" is very differently distributed throughout the year. In the summer quarter more than 14 per centum of the deaths yearly referable to this disease are recorded in Dublin, and more than 15 per centum in London. In the first quarter the figures are—Dublin, 31 per centum; London, 31 per centum; in the second quarter they are —Dublin, 30 per centum; London, 26 per centum; in the fourth quarter they are—Dublin, 24 per centum; London, 28 per centum.

From these numerical results it therefore appears that the fatality and (indirectly) the prevalence of pneumonic fever from season to season do not correspond with the seasonal prevalence and fatality of bronchitis. The latter disease—be it of primary or secondary origin—increases and kills in direct relation to the setting in of cold weather, with excessive relative humidity and increased and frequent precipitation in the form of rain, snow or sleet, and hail. It subsides in prevalence and fatality with the advance of spring and the advent of summer.

Pneumonic fever, on the other hand, increases less quickly in winter, and remains more prevalent and fatal in spring and summer than bronchitis; its maximal incidence coincides with the season of dry, harsh winds and hot sunshine in spring, when also the relative humidity is low, precipitation is scanty, while the diurnal range of temperature is extreme.

5. *Intemperance* is generally credited with increasing the predisposition to pneumonia. It certainly increases the risk to life caused by this disease. Dr. Hector Mackenzie, however, states that it has not been clearly proved that alcoholism makes an individual more susceptible to pneumonia. There was a clear history of alcoholism in about 8 per cent of 1392 cases treated at St. Thomas's Hospital, and in 9·8 per cent of the cases reported on by the Collective Investigation Committee of the British Medical Association.

6. *Occupation.*—In a letter to the Registrar-General of Births, Deaths, and Marriages in England, on the mortality of males engaged in certain occupations in the three years 1890-92, Dr. John Tatham, M.A., states that out of every 1000 deaths among males aged between 25 and 65 years, 107 were referred to "pneumonia." The "comparative mortality figure" for this disease varied from 257 for innkeepers in the industrial districts, 251 for general labourers in the same districts, 249 for coal-heavers, 248 for those employed in nail, anchor, chain, and other iron and steel manufactures, and 246 for hotel servants in London, to 45 for clergymen, 55 for lawyers, 93 for physicians, surgeons, and general practitioners, but only 32 for artisans, 36 for farmers, and 43 for schoolmasters. In the higher figures Dr. Arthur Newsholme thinks that the effects of dust and of alcoholism are probably combined.

Incubation

Definite references to the duration of the incubation period in pneumonic fever will be found at page 41 of a pamphlet on *Epidemic Pneumonia at Scotter*, by T. B. Franklin Eminson, published by H. Kimpton (London) in 1892; and in Dr. Parsons' report on the outbreak in question, published in the supplement to the *Twentieth Annual Report of the Local Government Board*, 1890-91 (pages 95 *et seq.* of the supplement).

Mr. Eminson says: "It is the general opinion that the incubation period is very short —a few hours to a few days—and this agrees with the experience of other epidemics; indeed, it has been said that there is frequently no incubation period properly so called, the patient falling ill very soon after exposure." Mr. Eminson narrates two cases in which the interval between exposure to the cause of the disease and its onset was 60 and 24 to 48 hours respectively. These periods, however, were only highly probable, by no means positively certain. Dr. Parsons says in his report: "The length of the period of incubation is doubtful, but it appears to be short, from a few hours to two days (assuming the disease to have been contracted at the time and in the manner locally supposed)." Dr. C. J. Russell M'Lean, in a paper on Epidemic Pneumonia at Yeadon, Yorkshire, in 1894, mentions three cases of pneumonia, in which a wife took

ill on the fourth day of her husband's illness, their son-in-law falling sick five days later; all three cases proved fatal. In December 1899 the writer met with a somewhat similar instance —a sister falling ill on the fourth or fifth day of her brother's attack, and both dying of epidemic pneumonia.

The following instructive series of cases was reported in *Lyon médical*, April 28, 1889:—A shopkeeper's child was convalescing from pneumonia when a servant boy developed the disease. He was removed to hospital, another boy taking his place, wearing the same suit of livery and sleeping in the same bed as his predecessor. Two days afterwards he sickened with pneumonia. A third boy was now engaged. He slept for two nights with the second boy, and thirty hours later fell ill of acute pneumonia.

It is interesting to observe that the life-history of the *Diplococcus pneumoniæ* of Fraenkel would almost preclude an incubation stage of more than five or six days at the outside. The virulence of this specific micro-organism, at all events when cultivated in solid media, is spent at the end of seven days, curiously enough the very period within which acute pneumonia is limited by its sharp and critical termination.

Clinical Features

In a large number of cases the prodromata and early symptoms of pneumonia differ in no way from those of any other infective fever. They are referable to the nervous system rather than to the lungs, and so betoken a general or constitutional poisoning as well as a mere local pulmonary infection.

In such instances the actual outburst of pneumonic fever is preceded by hours, or even days, of impaired health, signalised by languor, indigestion, loss of appetite and constipation, headache and dull pains in various parts of the body, alternate chills and flushes of heat, together with a general disinclination to work or amusement. To convey an idea of this vague departure from health the expressive though clumsy word "all-overishness" has been coined, but the state is better described by the more subtle French term "malaise." Andral relates a case in which the general symptoms of headache, debility, dulness of the intellectual faculties, flushed face, injected eyes, and frequent pulse appeared at least six days before the physical signs of pneumonia could be detected.

Not infrequently the onset of the disease may be stormy in its suddenness and impetuosity. A violent shivering fit, a racking headache and delirium, nausea and vomiting, a thickly coated tongue and foul breath, may, in such a case, suggest an attack of epidemic meningitis, or typhus, or acute gastritis, or enteric fever. All at once physical signs of a rapidly advancing consolidation of the lung—probably of the right apex—declare themselves, the general state of the patient simultaneously improves, and the attack may henceforth run a normal course.

But in more typical instances pneumonia is ushered in with sharp lancinating chest-pain, quickened, shallow breathing, cough, which is either constant or paroxysmal, hard and dry, as well as suppressed because of the pain it causes. This pain, when acute, is certainly often due to pleuritis, but when dull it may also indicate intense hyperæmia of the portion of lung which is the destined battlefield of the attack. Its usual situation is a limited area (which is also tender on pressure) near either nipple, but sometimes it is felt in the side (infra-axillary region), or in either hypochondrium, or even in the abdomen. In the last-named case it is very misleading, suggesting a gastric hepatitis or intestinal lesion rather than a lung attack. After a couple of days the pain subsides or disappears, as the movements of the affected lung gradually lessen, owing to consolidation due to coagulation of the exudation in the air cells.

Within the first few hours of the attack a very perceptible difference of movement on the affected and sound sides of the chest may be observed. The expansion lessens—it may be to vanishing point—on the affected side, whereas it increases considerably on the other, the sound lung working more and more energetically in response to the strain put upon it through the maiming of its fellow.

Many cases of acute pneumonia begin with a shivering fit or chills, which imply that toxins have already reached the central nervous system by absorption from microbic growth in the pulmonary air cells. The more intense this initial rigor the more deadly the poisoning. A sense of profound depression is apt to follow the shivering fit, or other nervous symptoms, such as headache, delirium, in children even convulsions or vomiting, with constipation or diarrhœa, quickly declare themselves. Another nerve symptom is the development of crops of herpes on or near the lips, the nostrils, the cheeks, the chin, or the jaws—this *herpes labialis* or *faciei* consists of groups of vesicles on a more or less hyperæmic base. The contents of the vesicles are at first serous, but may become purulent. They afterwards dry up and crust or scab, and the scabs finally fall off without leaving a scar. The development of herpes is usually looked upon as a favourable sign. It occurs in about two-thirds of the cases of acute pneumonia, but its apparent diagnostic value is minimised by its common appearance in simple fevers and ordinary catarrhal attacks. Geissler has estimated the mortality in herpetic cases at 9·3 per cent, in non-herpetic cases at 29·3 per cent.

The aspect of a patient in the early stage of pneumonia is flushed and feverish, often anxious. A deep hectic blush settles upon one or other

cheek—the "malar flush." The skin is dry and hot, almost burning the observer's hand with its pungent heat. This is the so-called *calor mordax*, common to pneumonia and scarlatina (Addison) and the hot stage of intermittent fever. According to Wunderlich, of

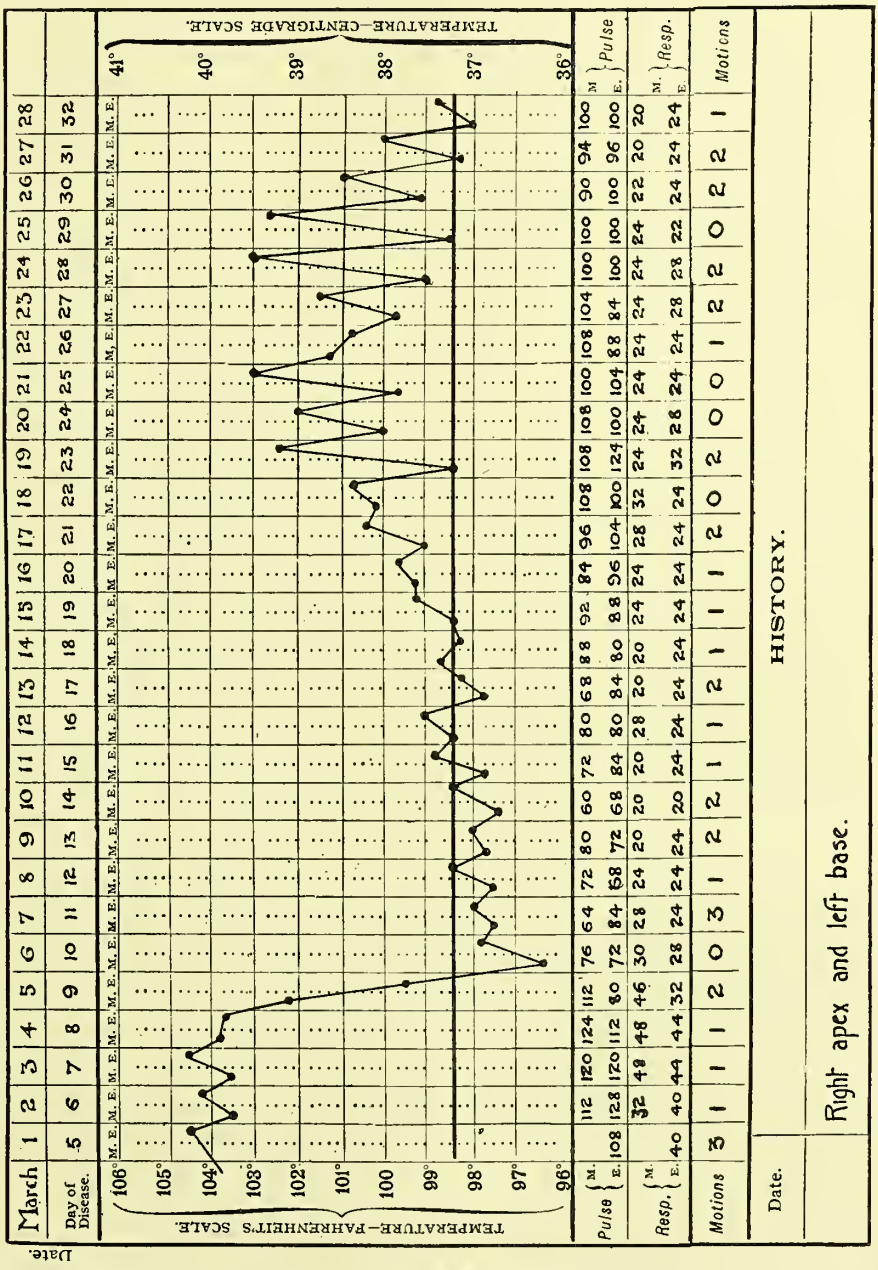


CHART 1.—Acute right apical pneumonia followed by a secondary septicæmic attack.

Leipzig, this pungent heat of skin belongs to those febrile diseases in which the high temperature depends chiefly upon increased production of heat, and in which the hand of the observer is therefore less able to place itself in equilibrium with the temperature of the patient's skin, because the continual over-production of body-heat (exalted thermogenesis) compensates for the heat lost by conduction. The relaxed or dilated state of the arterioles in pneumonia may also be a cause of *calor mordax*.

After an initial rigor or rigors, the thermometer rises briskly and in a few hours to 102° or upwards. It continues to rise until it reaches 104° F., or in severe cases 105·8° or more. For some days temperature hovers near these figures, according to the intensity of the case, showing, it may be, daily fluctuations of one or two degrees, the remissions occurring usually in the morning, the exacerbations in the afternoon or at night. Often a sudden and final rise of temperature to a fastigium immedi-

ately precedes the abrupt and critical defervescence so characteristic of pneumonic fever, and which may happen at any time between the fifth and eighth days of the attack. In other instances the fastigium is reached early, and a daily descent of temperature precedes the final crisis. Such is the continuous or subcontinuous type of pneumonic fever.

But other types present themselves, particularly in children, whilst the temperature chart is remittent or relapsing in character—a peculiarity very suggestive of a lobular rather than

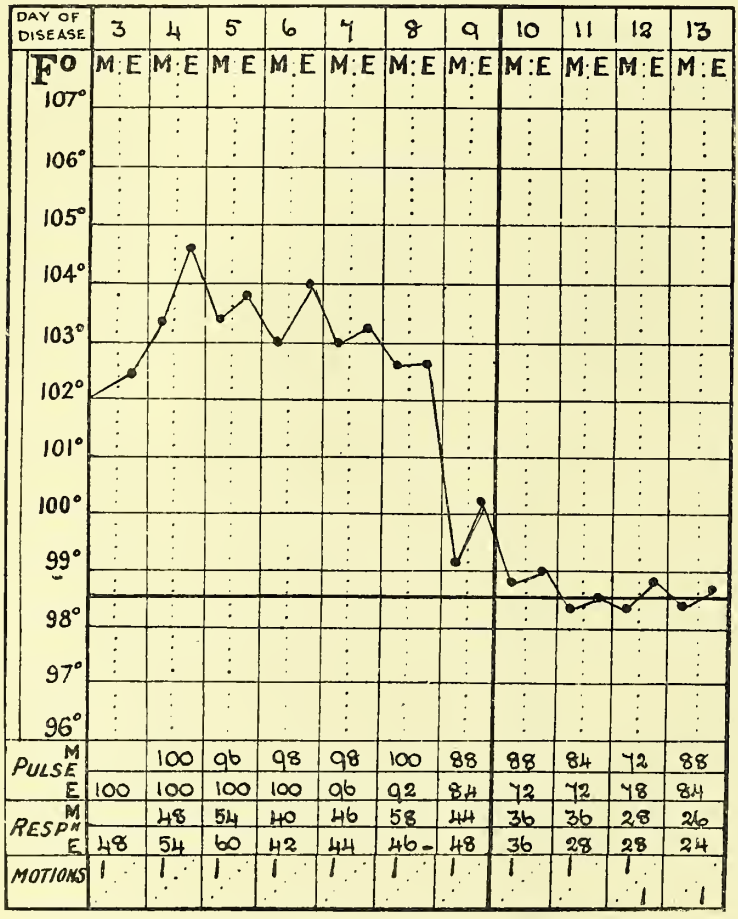


CHART 2.—Acute pneumonia terminating by crisis. Patient, a male aged 18.

a lobar pneumonia, or of what has been well called a *pneumonia migrans*, wandering pneumonia, which, erysipelas-like, invades region after region of the pulmonary parenchyma. In this form of the malady affected portions of lung clear up or resolve with marvellous rapidity, while other portions are passing into the earlier stages of hyperæmia, congestion, or consolidation, as the case may be. It is possible in such a case to study the physical signs of each and every stage of an acute pneumonia at once.

Very early in the attack the rate of breathing becomes quick, shallow, and laboured (*dyspnœa*). The respirations number 40 to 48, or even 60 or upwards, per minute, and this too when the pulse-rate may not exceed 120. The normal ratio of one act of respiration to four beats of the pulse is therefore lost in pneumonia, and so commonly as to constitute an important diagnostic sign of the disease. The difficulty of breathing is ever and anon increased by a short hacking cough, which the patient strives, though often in vain, to suppress because of the torture it causes by shaking the affected side, or by putting the affected lung and pleura on the stretch. At first the cough is dry, or unattended by expectoration. But, in most cases, sputa soon begin to come up, often with much difficulty and suffering. These sputa may be simply bronchitic in character, colourless, frothy, liquid, and consisting of mucus, epithelium, and young cells. Occasionally they at first consist almost entirely of pure blood. There is a true hæmoptysis.

Usually, however, there is no mistaking the expectoration of acute lobar pneumonia. It is rust-coloured, semi-clear or transparent, and so viscid as to cling to the bottom of the spitting-cup even when it is turned upside down. Examined microscopically this very characteristic sputum is found to consist of a network or mesh of coagulated fibrinous exudation, in which are entangled red blood corpuscles, giving it the rusty colour, altered epithelium from the air vessels and bronchioles, and many leucocytes. Such are the histological elements in the sputum of acute pneumonia of the croupous or fibrinous form. Occasionally the colour of the expectoration is greenish yellow instead of rusty. This is the so-called "greengage" sputum, because of its resemblance to the well-known preserve.

In certain unfavourable cases the sputa are thin and watery in consistence, dark-coloured, and offensive, resembling liquorice juice or prune juice. This is a bad sign. So also is a complete absence of expectoration, which may result from palsy of the bronchi and retention, or from the so-called "massive pneumonia," in which lung-cells and bronchioles alike are plugged with firm fibrinous coagula, which no effort of coughing is able to dislodge.

Coincidently with the beginning of the stage

of resolution the complete change. character, and its rusty colour gives place to yellow and finally white. It is coughed up more easily and in abundance. While yellow it is airless, sinks in water, and forms rounded, coin-like masses, whence it is called "nummular" (Lat. *nummus*, a coin). Finally, it becomes less and less purulent, and more frothy and mucoid. These changes are an outward sign of those which are taking place in the affected portion of lung. The fibrinous exudation has undergone fatty degeneration, has become emulsified, and is being in part absorbed, in part coughed up. Under the microscope the sputum in this stage is seen to consist of disintegrated young cells, fat granules, pigment, and fragments of fibrinous casts of the alveoli and terminal bronchioles.

In a final stage the sputum becomes a mere frothy mucus, such as is spit up in an ordinary bronchial catarrh.

During, and sometimes after the conclusion of, the pyrexial period, nervous symptoms, such as sleeplessness, delirium, and wandering may present themselves, particularly if the apex of the expectoration undergoes a It loses its sticky, viscid lung is the special seat of the disease, or if the patient has been intemperate. In the latter case an acute attack of delirium tremens may supervene. A certain amount of rambling of mind at night is common, but the appear-

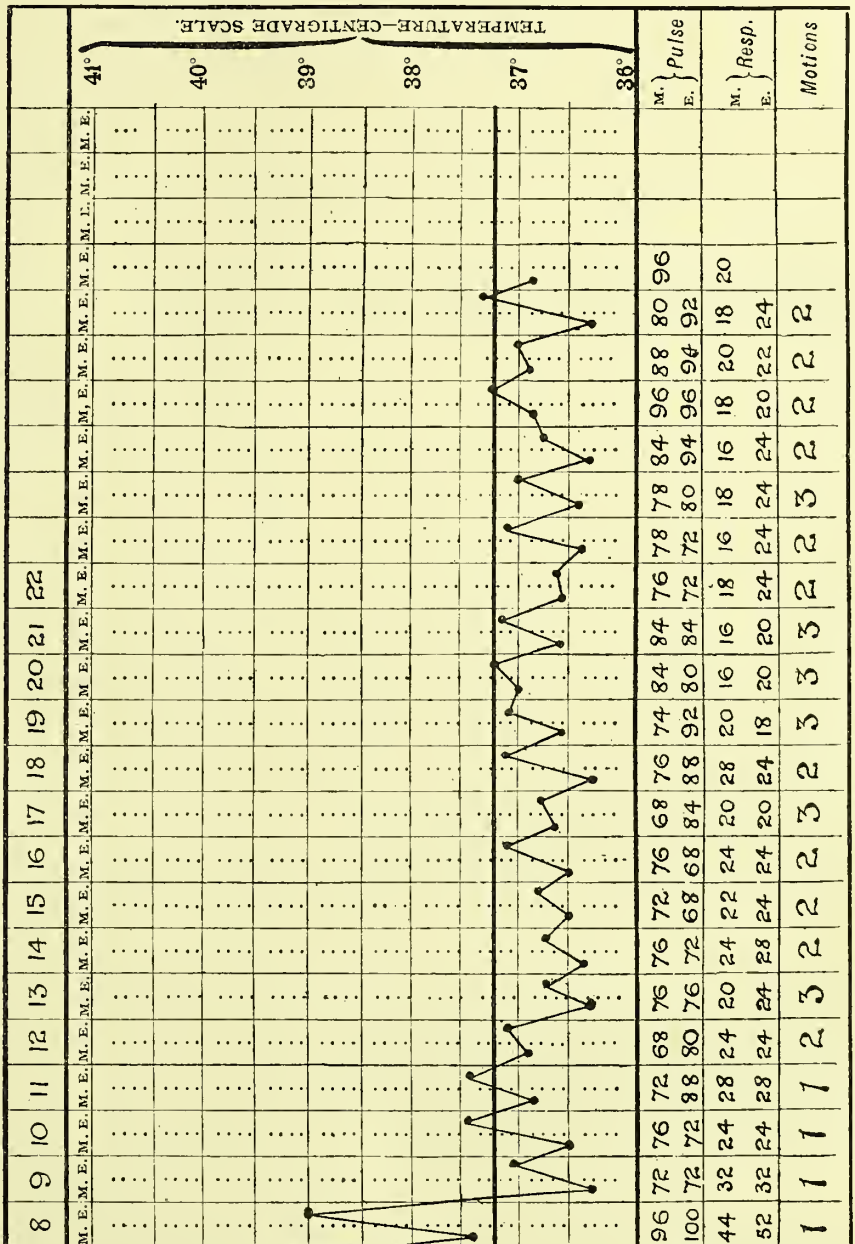


CHART 3.—Acute pneumonia terminating by halting crisis. Patient, a man aged 18. Right base affected.

ance of such a symptom by day is of the utmost import, and often foretells a fatal issue. On the whole there is less tendency in acute pneumonia to cerebral symptoms than in typhus or some of the other acute infections; the decided localisation of the poison in the lungs apparently relieves the nervous system, which often suffers severely when such a localisation does not take place or is of feeble intensity.

In a large number of cases pneumonia ends as suddenly as it began, so far at least as the

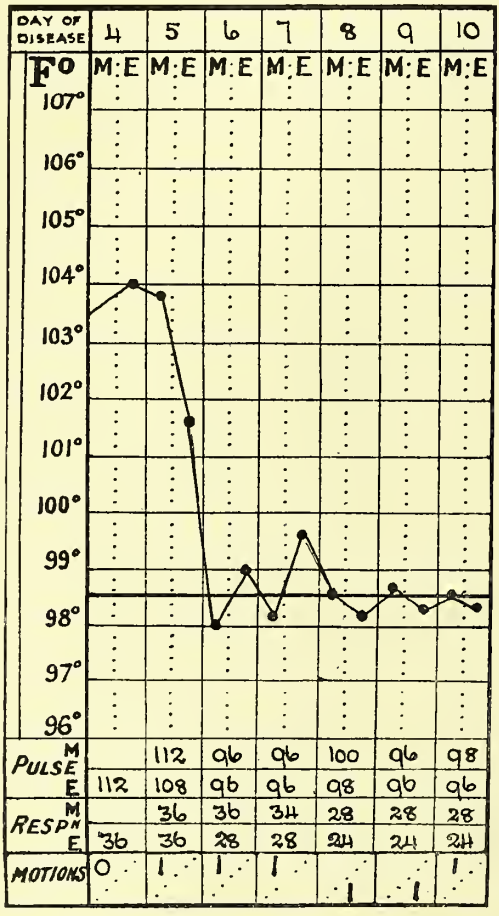


CHART 4.—Crisis in pneumonia on 5th day. Patient, a woman aged 22.

constitutional symptoms are concerned. The storm and stress of a tempestuous fever give place to the calm and comfort of returning health. So periodic and rapid is the defervescence, that of the pneumonic patient it may often be said that "at such an hour the fever left him." The physical signs, however, persist for a longer or shorter time after crisis, for the clearing or "resolution" of a consolidated lung or pulmonary lobe takes time. The fibrinous exudation undergoes a fatty degeneration, and at the same time liquefies. The resulting albuminous and fatty emulsion is then partly absorbed, partly expectorated.

Two notable departures from the cyclic course of pneumonia just described now and then present themselves. In a certain though small proportion of cases, after a threatening onset and one or two days of moderate, or it may be high fever, a sudden change for the better takes place, with a brisk fall of temperature, a moist perspiring skin, and a critical sleep. The attack has aborted. It is an example of *masked* or *larval* pneumonia. Professor Osler, of Baltimore, thus describes it: "A patient may have the initial symptoms of the disease, a slight chill, moderate fever, a few indefinite local signs, and herpes. The whole process may last for only two or three days; some authors recognise even a one-day pneumonia." In arriving at a diagnosis in such a case, the epidemic prevalence of the disease at the time, and the occurrence of undoubted pneumonia in the same house or family, should be taken into account.

To this mild and transitory form of the disease the phrase *ephemeral pneumonia* also has been applied.

The following example of masked or larval pneumonia recently came under the writer's notice:—

Staff-Sister Louisa C., aged 28 years, on duty at the Meath Hospital, Dublin, felt out of sorts and suffered from a cold with shortness of breath during the early days of March 1901. On Sunday, March 10, she became so poorly that she went to bed at 4 P.M. She complained of headache, malaise, shortness of breath, and a catch in her breathing. Next day she felt better, and so continued until Wednesday, March 13, on the morning of which day she awoke with headache, which got gradually worse until midday. At that hour (12 o'clock noon) she felt faint and had a severe rigor. The headache now became violent, and she suffered from increasing dyspnœa, so that she had to go to bed. Her temperature was now found to be 103° F. Next morning her aspect was typhus-like in the extreme—eyes suffused, pupils contracted, face dusky, eyelids heavy and drooping. P. 116, R. 22, T. 103°. Physical examination suggested a commencing right basic pneumonia. Towards evening the temperature fell to 99·4°, nor did it rise again, as the chart shows. The physical signs during the next few days were—a shade of dulness over the right base in succession to a remarkably clear percussion note; also increased vocal resonance, resembling pectoriloquy rather than bronchophony, and some crepitation (subcrepitus). The breathing was puerile over the left side. On Saturday, March 16, a patch of herpes labialis was observed near the left corner of the mouth.

In contrast to this—to quote Ernst Ziegler—it may happen that "during the course of an attack of pneumonia inflammation makes its appearance in other organs, such as the pericardium, meninges, and mediastinal tissue, the

mucosa and submucosa of the pharynx, soft palate, and nasal cavities, the conjunctiva, kidneys, etc., in which case the affected structure can be shown to contain micrococci." Such is complicated pneumonia—a condition which prolongs the fever movement, increases the risk to life, and may entirely upset the cyclic course of the disease.

In a paper on "Atypical Forms of Pneumonia," Dr. E. Palier describes a case of what he calls "Gastric Pneumonia," in which the symptoms closely resembled those of appendicitis. Abdominal section was performed by a consulting surgeon under the belief that disease of the appendix would be found. This, however, proved to be an error, and a post-mortem examination disclosed pneumonia associated with an empyema. Palier believes that abdominal symptoms in these cases of "gastric pneumonia" are to be ascribed to disturbance of the pneumogastric centre through the altered respiratory interchange. The abdominal pain is, in his opinion, analogous to the pain felt in the knee in hip-joint disease.

In March 1901, when epidemic pneumonia was rife in and near Dublin, the writer was called to see in consultation an anæmic girl, who for a week previously had been suffering from symptoms of acute gastritis, vomiting, pain and tenderness in the left hypochondrium, and diarrhœa. At first her tongue had been thickly coated with a loose white fur, but at the time of the consultation this had been shed and the tongue was red and raw. A few hours before the patient had got out of bed, when a sudden faintness came on. This was followed by a rigor, and her temperature rose rapidly to 105·2° with a small thready pulse of 140. Physical examination revealed dulness over the base of the left lung, subcrepitus and tubular breathing intermixed, bronchophony, and, in front, some pleural friction. In three days she was dead, the fatal issue being brought about by exhaustion induced by incessant vomiting, tachycardia, and extreme feebleness of the heart's action. In this case one had to choose between an acute pneumonia with such severe gastric symptoms as to mask that disease in its early stages, or a perforating gastric ulcer leading to a left basic pleuropneumonia. Unfortunately a post-mortem examination could not be obtained, but in favour of the latter diagnosis were the patient's age and sex, her anæmic state, and history of indigestion, and the sudden supervention of urgent gastric symptoms prior to the pneumonic rise of temperature and the development of the physical signs of an acute pleuropneumonia.

Had this patient been a child, a diagnosis of the "gastric pneumonia" described in 1880 by Adolf Baginsky, of Tübingen, might well have been made. In this form gastro-intestinal symptoms play a prominent part, while lung symptoms and signs do not obtrude themselves until just before crisis. Such cases may begin with vomiting, and diarrhœa is a conspicuous symptom.

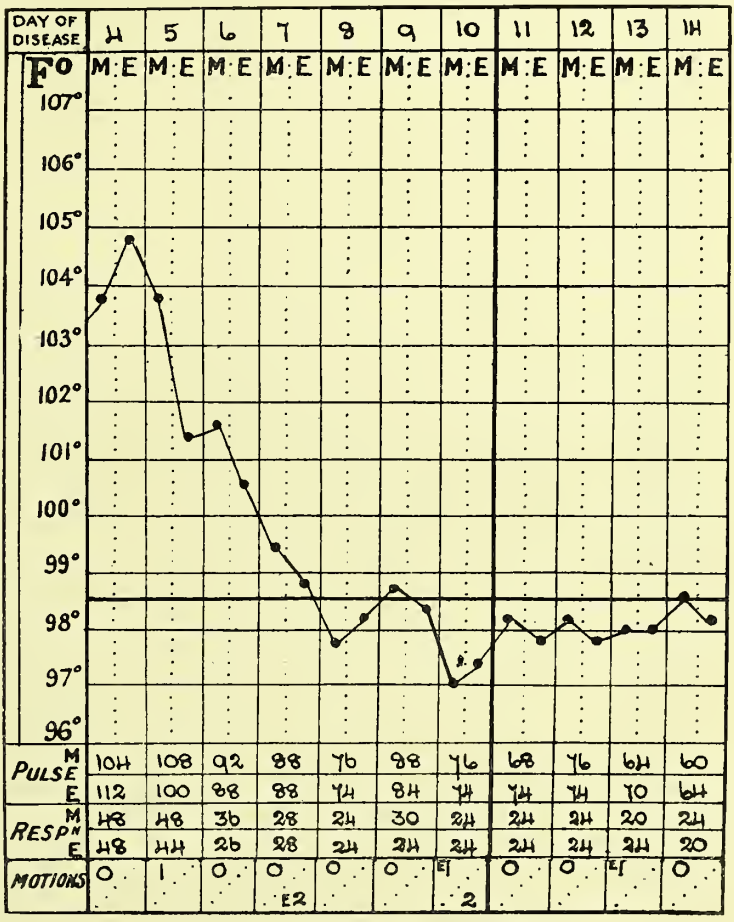


CHART 5.—Protracted crisis in acute pneumonia.

At the extremes of life also acute pneumonia shows striking atypical peculiarities which call for notice.

PNEUMONIA IN CHILDHOOD.—Apart from the fact that in very young children the exudation into the air vesicles is catarrhal rather than fibrinous, or croupous, in a large majority of cases, the clinical features of the disease are also often atypical. Gerhard, of Philadelphia,

long ago pointed out that broncho-pneumonia, or catarrhal pneumonia, was the form of pulmonary inflammation which chiefly occurred in children under five years of age, whereas lobar or fibrinous pneumonia was much more common in children aged between five and fifteen years. Among 98 cases of catarrhal pneumonia, von Ziemssen reports 67 as having occurred before the third year; Steffen 52 in 72 cases during the same period. "All observations agree," writes Jürgensen, "that the first three years of life are those which are most often attacked by catarrhal pneumonia." According to Osler, lobar pneumonia has been met with in the new-born, but he quotes Holt to show that of 270 cases of pneumonia in children under five years of age, 75 per cent were broncho-pneumonia. Notwithstanding this preponderance of a catarrhal exudation in the pneumonia of childhood, it is certain that the *Diplococcus pneumoniæ* of Fraenkel plays a part which is second to none in the etiology of infantile pneumonia.

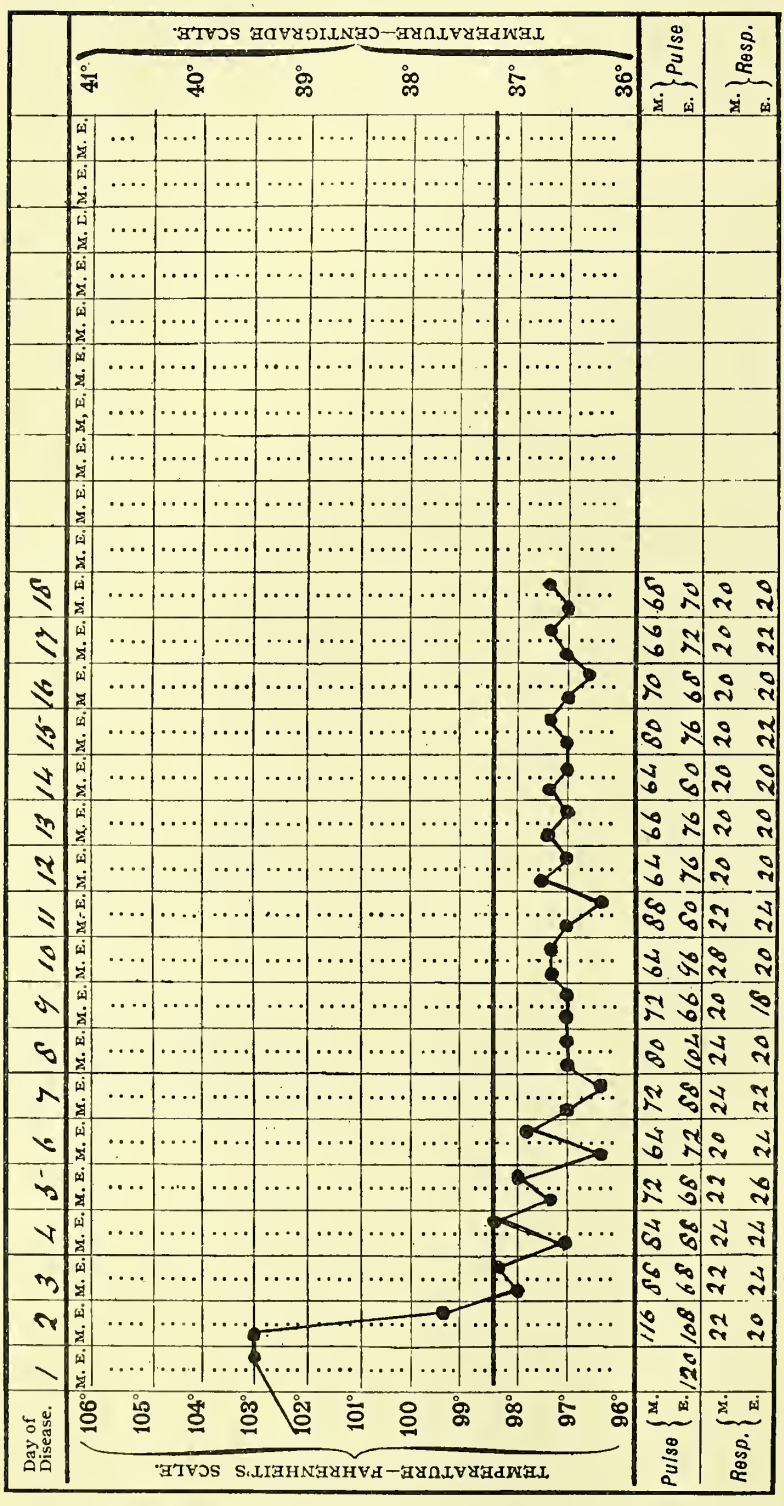


CHART 6.—Very early crisis (on the 2nd day) in acute pneumonia. Case of Staff-Sister Louisa C., aged 23 years, reported on p. 174.

The atypical symptoms and course of pneumonic fever in childhood add greatly to the difficulty of diagnosis. "Even a physician of limited experience," wrote Jürgensen in 1875, "can easily recognise pneumonia in adults, but in *young children* this is by no means an easy matter. Everyone who has seen the mistakes of his assistants in the practice of a large polyclinic will confirm this statement. Many children who are reported to have died from 'teething,' 'worms,' 'convulsions,' etc., have really gone to their graves with an undiagnosticated pneumonia."

Temperature usually runs especially high in patients under puberty—a fact which depends chiefly on the undeveloped mechanism of thermotaxis, or body-heat control, at tender

ages, but also on the greater frequency of apical pneumonia in very young subjects. Probably the occasional appearance of an erythematous blush upon the skin, simulating scarlatina, is due to paresis of the vaso-motor nerves. When a rigor, or more likely a convulsion, with vomiting, diarrhœa, redness of the fauces, and an erythema, usher in acute pneumonia in a child, a wrong diagnosis of scarlatina may be made, for which there is some justification in this remarkable grouping of symptoms. Cerebral symptoms often mask pneumonia in childhood. The attack may be ushered in by convulsions. Osler speaks of "the so-called cerebral pneumonias of children," in which the disease sets in with a convulsion, and there are high fever, headache, delirium, great irritability, muscular tremor, and perhaps retraction of the head and neck. The diagnosis of meningitis is usually made, and the local affection in the lung may be overlooked. Again, from anatomical considerations, even fibrinous pneumonia is apt to assume a lobular character. So vascular is the pulmonary parenchyma in childhood, and so fine and delicate are the connective tissue elements, that the spread of a pneumonia through contiguous lobules and even across the interlobar septa is an easy matter.

In children the adjacent serous membranes —pleura, pericardium, and endocardium—are very prone to infection by the *Diplococcus pneumoniæ* and other pneumonic micro-organisms. In the case of a little boy, aged 3½ years, recently under the writer's care, the disease almost spent itself in producing a pericarditis with endocarditis. Only as an afterthought, as it were, did the physical signs of a left basic pneumonia show themselves.

A difficulty may arise in another way also from the circumstance that pleurisy with effusion may in very early life closely simulate pneumonia, not only in its fulminant clinical course with fever, cough, and dyspnœa, but also in its physical signs. Even in adult cases the blocking of a system of bronchial tubes by plastic exudation may interfere with the development of the classical signs of pneumonic consolidation — bronchophony, tubular breathing, and increased vocal fremitus. In such a case the dulness on percussion and absence of breath sounds—natural or adventitious—may be attributed to a pleural effusion rather than to a pulmonary consolidation.

Young children expectorate indeed, but do not *spit out*—the expectoration is commonly swallowed. This removes from our ken one easy means of diagnosis in pneumonic fever—the inspection of the rusty viscid sputum. But it does more than this by contributing to gastro-enteric derangement, the toxin-laden sputum when swallowed probably acting as a local irritant in the stomach and intestines. Hence vomiting and diarrhœa, tympanites, abdominal tenderness and pain, possibly convulsions or other cerebro-spinal symptoms.

Reference has already been made to the form of acute pneumonia in children to which Baginsky, of Tübingen, gave the name of "gastric pneumonia" in 1880.

When an attack of vomiting takes place, characteristic rusty sputa may be seen in the ejected matters, confirming the diagnosis of a croupous pneumonia, and proving that the expectorated phlegm has been swallowed in the first instance. As a diagnostic, E. Henoch lays stress on the character of the breathing, which is short, very rapid, with *grunting expiration.*

The pneumonia of childhood often runs a short course and terminates in recovery. Allusion has already been made to the remittent and relapsing character of the pyrexia in young children, a range of temperature which suggests a lobular distribution of the pulmonary trouble.

The danger to life from croupous pneumonia occurring in previously healthy children is trifling. Feeble children, however, succumb to the disease as easily as patients of more advanced age (Jürgensen). Henoch lost only 7 out of 64 cases in young children. Out of 201 cases in children under 16 years—the great majority being under 10 years—von Ziemssen lost only 7 (3·3 per cent). At the Kiel Policlinic, Jürgensen lost only 4 out of 110 patients under 10 years of age. Barthez lost only 2 out of 212 children aged from 2 to 15 years. In striking contrast to these figures is the heavy death-toll which catarrhal pneumonia exacts from its subjects of tender age. Bartels lost all his patients under 1 year of age; after that period a little more than one-third. Von Ziemssen lost just one-half of the children under 1 year, about 38 per cent (17 out of 45) of those from 1 to 3 years old, only 25 per cent (8 out of 31) of those above 3 years of age.

Pneumonia in Old Age. — Insidious in its onset, and it may be latent in its course, pneumonia is a frequent and perilous disease of advanced life. By it in many a case the silver cord is loosed, when already strained almost to breaking point by the presence of some other and older malady, such as carcinoma, system diseases of the brain and spinal cord, chronic nephritis, gout, or other dyscrasia. The weak and degenerated heart of old age cannot battle against the added strain of a pneumonic fever and pulmonary consolidation, and so pneumonia is rightly dreaded by all physicians when it supervenes in the aged and infirm.

In its clinical features also the pneumonia of advanced life differs from the typical disease. Its seat is the most dependent parts of the lungs, whence perhaps it has some claim to the term "hypostatic pneumonia." It is less febrile, less acute, less lobar in its distribution. The sputum is less rusty, more cellular, than that of ordinary acute pneumonia, and so the attack works its

treacherous way to the fatal end. During the "age of decline," as Jürgensen observes, pneumonia is one of the most dangerous diseases. The greater the wear and tear of the body (he adds) the more fatal the disease becomes. At the Greifswald Policlinic in ten years there were 941 cases of pneumonia with 102 deaths (= 10·8 per cent). From 40 to 50 years of age the mortality was 9·5 per cent, from 50 to 60 years, 20·0 per cent, from 60 to 70 years, 37·5 per cent.

If we inquire more closely how death is brought about in pneumonic fever, we must agree with Jürgensen when he says: "It is the heart, and always the heart, upon which the burden is ultimately thrown"; and again: "The danger in croupous pneumonia threatens principally the heart of the patient. Death results from insufficiency of the heart." If this proposition be accepted as proved, we see why old people so readily succumb to this disease. The muscle of their heart is either wasted (simple atrophy), or it is fattily degenerated or infiltrated with fat, and so when the added strain of a continued fever and of a sudden extreme impediment in the lesser circulation is put upon it, the heart fails and death results.

Hence the necessity for daily, even hourly, observations on the heart's action and pulse during a pneumonia. The quality of the pulse as regards strength and volume and tension is of more importance than its mere frequency, and this applies to the cardiac impulse also. Undue rapidity of the heart and pulse-beat is unfavourable; still more so is any irregularity in volume or rhythm of the arterial throb, or an intermission in the beat. It is to be remembered that in acute pneumonia the normal ratio between the pulse and respirations is disturbed. That ratio in health is 4 to 1, perhaps more strictly 9 to 2. In pneumonia it may become only 3 to 1, or perhaps only 2 to 1. "In severe cases the frequency of the respiration approaches that of the pulse; the former may equal, and even exceed, the latter." These are Jürgensen's words, and he adds: "Such cases, however, are very rare, but I have met with them, especially when the arteries were in a high degree atheromatous."

This disproportion between the pulse-rate and the respiration is diagnostic of pneumonia, for it fails to present itself in any other febrile disease except, perhaps, cerebro-spinal meningitis in its earlier stage.

Physical Signs

In 1896 Dr. Richard H. Kennan drew attention to the myotatic irritability of the pectoral muscles observed in acute croupous pneumonia, and expressed the opinion that the condition was a valuable early diagnostic sign of the disease—the more so as the myoidema is confined to the affected side. The writer has had many opportunities of confirming Dr. Kennan's observation, and can testify to the value of the sign. To elicit the sign Dr. Kennan recommends that a finger of the left hand should be placed flat upon the chest in the direction of the course of the fibres of the pectoralis major. The pleximeter thus formed should be sharply percussed by the middle finger of the right hand, used as a plessor, when a rapid contraction immediately succeeding will be felt in the muscle under the finger of the left hand. Dr. Kennan claims for this sign that it may precede the earliest inspiratory crepitations of pneumonia.

It will be convenient to analyse the physical signs which show themselves in the several stages through which the disease usually passes, namely—the stage of fluxion, or the preliminary stage described by William Stokes, the stage of engorgement or congestion (*engouement* of French writers), the stage of fibrinous exudation, or of red hepatisation or consolidation, and the stage of grey or yellow hepatisation or of resolution.

1. *Preliminary Stage of Stokes.*—The affected side shows lessened expansion and myotatic irritability. The percussion note is hyperresonant, or tympanitic, from relaxation of the lung tissue; or there may be a commencing dulness in patches. The breath sounds are harsh and rough, and there is already some increase in vocal resonance.

2. *Stage of Engorgement.*—There is lessened expansion. On palpation vocal fremitus is increased. The percussion note is abnormally clear, or even tympanitic in places, deficient in resonance in other places. The vesicular murmur is harsh, but at the same time weak. Towards the end of inspiration a fine crepitation is heard over the affected lobe. This is the *râle crepitant* of Laennec, to whom we owe so much of the nomenclature of diseases of the chest. Laennec compared this fine crepitus to the crackling of salt thrown upon a fire, but Dr. C. J. B. Williams made a more apt comparison when he likened it to the sound produced by rubbing one's own hair close to the ear between the fingers. Dr. Octavius Sturges imitated the crepitus of early pneumonia by the following manœuvre: A fine sponge, dipped into a weak solution of gum, is lightly compressed within the hand; upon the gradual relaxation of the pressure a crepitus ensues, which will be more or less fine with the degree of dilution of the gum solution.

These comparisons are but of little value. The sound is one *sui generis*, once heard never to be forgotten—it is pneumonic crepitation.

3. *Red Hepatisation.* — In this stage the affected side may be slightly enlarged, especially in young patients, whose chest walls will more readily yield to pressure than the "rigid thorax" (Freund) of old age. Expansion is now markedly lessened. Tactile or vocal fremitus is equally increased, provided always that the bronchi communicating with the solidified part

of the lung are not blocked, or that a pleural effusion is not already taking place. Dulness on percussion is now elicited over the solidified lung, but the note is not flat or absolutely dull, as in pleural effusion, but hollow, tubular, or perhaps slightly amphoric or metallic. Just before consolidation takes place the cracked-pot percussion sound (*bruit de pot fêlé* of Laennec) may be present.

Associated with dulness on percussion, a sensation of increased resistance is conveyed to the observer's hand when the chest wall is struck over the solid lung. The air-containing vesicles of the healthy lung rebound to the percussion blow, the blocked-up vesicles of the consolidated lung do not—hence the feeling of resistance, which is in proportion to the dulness on percussion.

Fine crepitation is still heard along the confines of the consolidated area, but for the most part the breath sounds now are bronchial or tubular — blowing, whiffing, or it may be metallic in character, unless the tubes are blocked. Vocal resonance is increased, assuming one or other of the characters described as pectoriloquy (laryngeal or tracheal), bronchophony (bronchial), or ægophony (bleating bronchophony). The last-named variety indicates interference with the transmission of the vocal vibrations by the presence of fluid in the pleura. For two reasons the heart sounds are intensified over the area of solidified lung: first, the absence of the normal vesicular breathing throws them into relief; secondly, the solid lung acts as a conductor.

4. *Resolution.*—In this final stage the dulness on percussion may subside in patches, or generally, and in a such a case with singular rapidity. At the same time a new crepitation is heard, particularly when the patient draws a deep breath. It is not so fine as the viscid crepitus of the earlier stage, and may be described as subcrepitant or medium—that is to say, the râles occupy an intermediate position between fine crepitus and coarse or mucous râle. This subcrepitus indicates the reopening of the air vesicles and the readmission of air, once the altered and emulsified exudation has been expectorated or absorbed. The expansile movements of the affected side of the chest gradually increase. At first they are voluntarily limited by the patient (vital ankylosis), because the freer movements put on the stretch any pleural adhesions which may have formed and so cause pain. Indeed, both after pleurisy and after pneumonia a temporary increase of that pain which is so well described as a "stitch in the side" is commonly observed as a result of the freer expansion of the chest wall.

Complications

"During the course of an attack of pneumonia," writes Ernst Ziegler, "inflammation makes its appearance in other organs, such as the pericardium, meninges, and mediastinal tissue, the mucosa and sub-mucosa of the pharynx, soft palate and nasal cavities, the conjunctiva, kidneys, etc., in which case the affected structures can be shown to contain micrococci." In this sentence we have primary infections by the *Diplococcus pneumoniæ* indicated as a cause of complications. But there may be numerous secondary infections also by other pathogenic micro-organisms than those peculiar to pneumonia; and, in the third place, the marked leucocytosis in pneumonia, and other blood alterations, may give rise to complications. The white blood corpuscles increase from 10,000 to 19,000 or more per cubic millimetre; as many as 68,000 have been found. The increase is generally in the polymorphonuclear cells (Stengel). Fibrin is increased from 4 to 10 parts per 1000, so that a condition of marked hyperinosis exists.

One of the most frequent complications of acute pneumonia is *pleuritis.* In most cases the pleura is more or less involved in the morbid changes, so that the name *pleuropneumonia* is appropriate. In a comparatively small number of cases the affection of the pleura becomes so acute and widespread as to constitute danger to life. Then it becomes a complication. This happens in from 5 to 15 per cent of the cases of pneumonic fever. Usually the amount of effusion is small, but owing to the consolidation of the lung a comparatively scanty effusion may make a great show. The solid lung cannot collapse, and so the effused fluid passes upwards above the solidified lobe, which it also surrounds. The effusion may be fibro-serous, sero-fibrinous, or purulent (*empyema*). This complication is especially apt to occur in childhood, at which period of life the effusion is very commonly purulent from the outset.

In the aged, and also in young children, *bronchitis* plays a prominent part in the symptomatology of acute pneumonia, even when the exudation into the air vesicles is fibrinous or croupous. Just as there is in one direction a certain amount of pleuritis in nearly all cases of pneumonic fever, so also there is almost always more or less bronchial catarrh in the other direction. This, however, does not count as a complication unless it is so widespread as of itself to cause risk to life through palsy of the bronchial muscle in the aged, or mechanically in children by blocking of the bronchioles by plugs of coagulated fibrinous exudation (plastic or croupous bronchitis). Acute capillary bronchitis, according to Magnus Huss, was present in 140 out of 2616 cases of croupous pneumonia observed in Stockholm—that is, in 5·4 per cent of the patients (Jürgensen).

Pericarditis and *endocarditis* are rare but perilous complications, particularly in cases in which the heart muscle is implicated, in myo-

carditis, or in hyaline or cloudy degeneration. Pericarditis may be the direct outcome of a bacterial infection, or may arise from an extension of inflammation from the neighbouring pleura to the pericardium by continuity. Endocarditis takes its origin in a bacterial infection, which may be either primary (diplococcic or pnemonococcic) or secondary (streptococcic or staphylococcic). In the latter case the attack may assume the terrible characters of malignant or infective endocarditis, with early death. Andrew H. Smith says that pericarditis occurs in from 2 to 8 per cent of the cases of pneumonia. At the Presbyterian Hospital, New York, it was made out during life in 6 out of 485 cases. He shows that it is often overlooked during life, the area of the pneumonic dulness including that of the pericardial effusion, and auscultation not being practised at the time when the pericardial friction sound might have been heard. At Vienna there were 27 instances of pericarditis among 5738 pneumonia patients, or one-half (0·5) per cent.

Collateral hyperæmia, *gangrene of the lung*, and *abscess of the lung* must be mentioned as occasional complications intimately related to the primary lung affection. The first is brought about in part by the action of the right ventricle in driving the whole blood-stream through the non-consolidated parts of the lungs. "The second factor," says Dr. Andrew Smith, "is the aspiration of an excess of blood into the sound lung by the action of the muscles of respiration. The inspiratory effort, not resulting in adequate expansion of the crippled lung, produces a condition of negative pressure in the sound one, and blood flows into the latter in consequence."

When the nutrient blood-vessels of the lung become blocked from thrombosis or by embolism, and saprophytic or putrefactive bacteria are present, the serious condition known as pulmonary gangrene results. Its occurrence is denoted by the stench from the breath and the horribly offensive sputa which the patient brings up, with much suffering in some cases. These sputa will be found on microscopic examination to contain strands of yellow elastic tissue—a proof that the lung structure is breaking up.

"In rare instances," writes Dr. Andrew Smith, "the affected portion of the lung undergoes a suppurative process. The surface has then a yellowish colour, and on section a purulent fluid exudes. The cells, resembling pus cells in all respects, not only fill the alveoli, but infiltrate the interalveolar walls. The pressure thus induced interferes with the nutrition of the septa, and may lead to their softening and breaking down. This may result in the formation of abscesses; but the condition, if at all extreme, usually proves fatal before this point is attained. Abscess of the lung, as an event of pneumonia, occurs in between 1 and 2 per cent of all cases. The abscess may have firm walls, or may be only an irregular cavity in broken-down tissue. Abscesses vary in size from that of a pea to the dimensions of an entire lobe. They sometimes discharge through a bronchus; at other times, when small, they become encapsulated and undergo caseation." Traube first called attention to a change in the colour of the sputum to *green* as a diagnostic of suppuration in the lung.

Jaundice is an occasional complication of pneumonia. It occurred in 30 out of 5738 cases at Vienna (0·6 per cent), in 23 out of 2616 cases at Stockholm (0·9 per cent), but in 65 out of 230 cases at Basle (28·3 per cent), according to Fismer. Probably the last-mentioned high percentage included slight degrees of icterus, the result of hepatic congestion. Intense icterus is caused by a duodenal catarrh leading to obstruction of the bile ducts. Lastly and rarely, there may be a dangerous form of non-obstructive jaundice due to profound blood changes. G. Mante has shown that this variety depends on the hæmatolytic action of the *Diplococcus pneumoniæ lanceolatus*.

Parotitis is a very rare but an exceedingly serious complication, which is met with in aged or debilitated patients. It is usually unilateral, according to Grisolle, and generally terminates in suppuration or even in gangrene. In the reports of the Vienna hospitals this inflammation of the parotid gland is mentioned only six times in 5738 cases. Jürgensen never saw this complication—a sufficient proof of its rarity. Testi in 1889 obtained the *Diplococcus pneumoniæ* in pure cultures from the pus in a case of parotitis complicating acute croupous pneumonia.

Meningitis may be caused by the *Diplococcus pneumoniæ* directly, or — as pointed out by Jürgensen — the presence of croupous pneumonia is a powerful predisponent to epidemic cerebro-spinal meningitis. Of course, when Jürgensen wrote, the *Diplococcus intracellularis meningitidis* of Weichselbaum had not been isolated. The truth seems to be that the meningeal diplococcus undoubtedly sometimes gives rise to a pneumonia, while the pneumonic diplococcus with equal certainty may light up a meningitis.

Among other complications may be named *otitis media*, the purulent form of which may reinfect the lung; thrombosis of the femoral vein, usually the left; and various paralyses (including peripheral neuritis), which are, however, infrequent. *Diarrhœa* is also occasionally observed. The rarer sequelæ of pneumonia are: (1) Abscess of the lung; (2) pulmonary gangrene, either circumscribed or diffuse; (3) caseation, or "cheesy infiltration," in which a secondary tubercular infection often plays an important part; and (4) fibrosis, or fibroid induration of the lung (chronic interstitial pneumonia, the "cirrhosis of the lung" of Sir Dominic Corrigan).

Pulmonary consumption very rarely follows in the wake of croupous pneumonia, whereas it is a common sequela of catarrhal pneumonia. In a draper's assistant, a young man aged 19, lately under the writer's care, a suspicious secondary fever set in after eight or nine days' pyrexia. Repeated examinations of the sputum failed to show the presence of tubercle bacilli, and the disturbance passed off after a fortnight, leaving no ill effects. The attack was probably due to some septic absorption in the resolution stage of a right apical pneumonia (see Temperature Chart I., p. 171).

Diagnosis

In a well-marked typical case the diagnosis of acute croupous pneumonia presents no difficulty, at least after a day or two. But in masked or larval cases, or where the disease runs an atypical and sluggish course, the diagnosis may be attended with much doubt. Again, there are morbid states of the lung, which in their symptoms and physical signs may simulate acute pneumonia.

Typhus.—The onset of pneumonia often closely resembles typhus in its pre-eruptive stage. The initial rigors, headache, sleeplessness, brisk rise of temperature, constipation, and suffused eyes and face, are common to both diseases. The diagnosis is settled by the respiratory symptoms of the lung attack, its rusty sputum, and its physical signs. Also the epidemic prevalence of either one or other disease should be taken into account.

Enteric Fever.—In the Annual Report of the Metropolitan Asylums Board for 1897, it is stated that of 293 patients admitted to hospital certified as suffering from enteric fever, 4 had bronchitis and 61 had lobar pneumonia. These figures would lead to the conclusion that enteric fever and acute pneumonia are often confounded with each other. The mistake might be excusable in those rare instances in which pneumonia runs a protracted course, with continued fever, and perhaps diarrhœa and wasting. But at the onset of an attack a systematic examination of the chest should obviate a wrong diagnosis.

Even when cough and expectoration are absent, the rapid respiration, unequal expansion of one side of the chest, and the results of a careful physical examination, should clear up the diagnosis, particularly if the Widal test yielded a negative result. Of course no reference is here made to the so-called typhoid or ataxic state, which may supervene in either enteric fever or pneumonia.

Meningitis.—Dr. Osler, writing in 1890, observes that "nervous symptoms are more frequent in pneumonia than in typhoid, and from the onset may so dominate that the local lesion is entirely overlooked. For instance, in the case of cerebral pneumonia of children, in which the disease sets in with a convulsion, there are high fever, delirium, great irritability, muscular tremor, and perhaps retraction of the head and neck, and consequently meningitis is usually diagnosed. Cases occur in which the malady sets in with acute mania. For example, a young man behaved so strangely on the train that he was handed over to the police as a lunatic, and as he had no cough and little fever (though he complained of a pain in the side) pneumonia was not recognised for several days. Again, pulmonary features are frequently masked where the patient has delirium tremens, and error is certain to occur unless it is made an invariable rule to examine the chest in these cases." He goes on to say: "There are cases with toxic features resembling uræmia; without chill, cough, or pain in the side the patient may develop fever and a little shortness of breath, and then gradually grow dull and heavy, and within three days there may be a condition of profound toxæmia, with low, muttering delirium. In many of these cases the most characteristic symptoms of the disease may be absent, particularly the cough and the rusty sputum; but the physical signs, if they are elicitable, are well marked. Even in the gravest of these cerebral cases the crisis and the onset of convalescence may occur in the ordinary way, and the patient may pass from a condition of extreme danger to one of perfect safety."

Other Chest Affections.—When in catarrhal pneumonia many adjacent lobules are affected, the physical signs of consolidation of the lung may be present. But the history of the attack should solve the question of diagnosis, although the distinction between catarrhal and croupous pneumonia may not matter much so far as treatment is concerned.

Passive or mechanical congestion of the lungs, leading to pulmonary œdema or hypostatic consolidation, may resemble pneumonia in regard to physical signs. But the conditions named develop slowly, are for the most part afebrile, and are neither unilateral nor lobar in their distribution.

An apical pneumonia may excite fears of a pulmonary tuberculosis, which even the failure to discover Koch's bacillus in the sputum may not lull to rest. In a recent instance the writer was very uneasy about a patient who, when recovering from a sharp attack of right apical pneumonia, developed a secondary fever of some ten days' duration, at the same time assuming a very phthisical aspect. Koch's bacilli were not found in the sputum, and the patient completely recovered. This secondary fever appeared to be due to absorption of some toxic agent in the exudation while undergoing resolution. The clinical chart in this case is given on p. 171.

But it is with an acute *pleurisy* with effusion that pneumonia is most likely to be confounded, particularly in children. The onset of pleurisy may be sudden, with shivering, pain in the side,

rapid and shallow breathing, with cough and fever. But the temperature does not run as high as in pneumonia, and the other evidences of a profound intoxication are wanting. The character of the cough is dry, short, sharp, and hacking in pleurisy, and the course of the attack is less cyclic than pneumonia. The sputa are indicative of catarrh or of pulmonary œdema in pleuritis.

Great attention must be paid to the physical signs, which in most cases afford grounds for a differential diagnosis. In the first place, on inspection, the natural furrows of the intercostal spaces are maintained in pneumonia, whereas in acute pleurisy with effusion they are effaced, the spaces sometimes bulging. On palpation, vocal or pectoral fremitus is usually exaggerated in pneumonia, lessened or lost in pleurisy. The cardiac impulse may be felt in or very near its usual situation in pneumonia; it may be widely displaced in pleurisy. On percussion the absolute dulness (amounting to flatness) of pleural effusion contrasts with the relative dulness of a pneumonic consolidation. And, lastly, on auscultation, we have in order in pneumonia rough breath sounds, fine crepitation, tubular breathing, and bronchophony or ægophony, with crepitus redux in the resolution stage. In pleuritis there are—in order—friction sounds, faintness or absence of breath sounds, or distant rhonchi and râles, with lessened vocal resonance. Signs of displacement of organs, namely, the heart, liver, spleen, and stomach, are present in cases of extensive pleural effusion.

Unfortunately, the principal signs may help but little in the case of young children, but in them pneumonia still generally keeps its cyclic and periodic character—the duration of the attack being numbered by days instead of weeks, as in pleurisy.

Prognosis

Pneumonia is always a serious disease, for its presence means interference with respiration and circulation alike. Yet the death-rate is not excessive in the United Kingdom.

It has already been shown that the mortality per cent of those attacked equals 14·5 in England and Wales. In the United States of America pneumonia appears to be a far deadlier disease than in the United Kingdom. According to Osler, pneumonia is the most widespread and the most fatal of all acute diseases. In the United States, during the census year 1890, 76,496 persons died of it, a death-rate per 100,000 of population of 186·94. "More deaths are attributed to it than to any single form of disease, except consumption" (Census Report). Hospital statistics, quoted by Osler, show a very high rate of mortality—for example, Montreal General Hospital, 20·4 per cent; Johns Hopkins Hospital, 29·8 per cent; Pennsylvania Hospital, 29 per cent; Boston City Hospital, 29·1 per cent; and highest of all, in a southern climate—Charity Hospital, New Orleans, 38·01 per cent. Osler quotes Wells as collecting from various sources 223,730 cases, with 40,276 deaths, a mortality of 18·1 per cent.

The fatality of pneumonia is governed by the patient's age, sex, habits, previous state of health, the seat and extent of the lung affection, and the epidemic virulence of the disease for the time being.

Age.—Croupous pneumonia is not fatal to young children, or to previously healthy, sober adults. It is extremely deadly to old people, and in the presence of a broken-down constitution. "The greater the wear and tear of the body, the more fatal the disease becomes" (Jürgensen).

Sex.—Less common among females, pneumonia is more fatal to them than to males. Of 223 cases treated in the Presbyterian Hospital, New York, 170 were males, with a death-rate of 28·8 per cent; and 53 were females, with a death-rate of 31·2 per cent (Andrew H. Smith). One reason for the higher mortality among women is that the condition of pregnancy adds immensely to the danger of pneumonia, owing to the hyperinotic state of the blood which attends pregnancy.

Habits.—Alcoholic excess enormously adds to the peril of pneumonia. Among 428 cases admitted to the Presbyterian Hospital, New York, the mortality was 20 per cent among the non-alcoholic, 32 per cent among the moderately alcoholic, but 70 per cent among the markedly alcoholic (A. H. Smith).

Previous Ill-Health.—Damaged kidneys, of course, play an important rôle in increasing the mortality among alcoholic pneumonia patients. Rheumatism, malarial poisoning, diabetes, chronic nephritis, heart or lung disease, especially phthisis, all influence the prognosis unfavourably.

Situation and Extent.—An apical pneumonia is more dangerous than a basic attack, because it less readily resolves. Double pneumonia is very dangerous. So is an attack which involves the whole of one lung, or is accompanied by a croupous bronchitis (massive pneumonia). Right pneumonia is stated to be more fatal than left.

Epidemic Influences.—At times of epidemic prevalence the disease is apt to become virulent. The pneumonia of influenza is one of the deadliest of diseases, directly because it tends to spread through the lungs, and is accompanied by a general bronchial catarrh; indirectly, because of the concomitant heart-palsy.

Among unfavourable prognostics are high fever (above 105° F.), very rapid respirations without hyperpyrexia, tachycardia, and feeble pulse, accentuation of the pulmonary second sound followed by loss of this character (implying weakening of the right ventricle), absence

of expectoration, or presence of prune-juice expectoration, marked cerebral symptoms, gastro-enteric disturbance, with diarrhœa and tympanites, (in women) pregnancy. Excessive leucocytosis indicates suppuration. Moderate leucocytosis is regarded as favourable, and so are local eruptions of herpes.

Treatment

In attempting to deal with this vast subject we propose to limit the scope of our inquiries by omitting the merely historical part of the question, and by boldly stating at the outset that the general principles of treatment of the infective fevers apply in their entirety to pneumonia fever also. This disease, in a word, like them, is an essential or constitutional malady, self-limited in duration. No doubt its normal course is often interfered with and interrupted by accidental complications or sequelæ, which may themselves demand vigorous treatment. But to "cure pneumonia" is still, malgré the brothers Klemperer and their serum treatment, a feat as impossible of achievement as to cure typhus fever. "We know of no cure for fever; no man has ever cured it," wrote William Stokes, of Dublin, many years ago. "It is, however, curable spontaneously. If you leave it to its own course it is capable of curing itself. It will spontaneously subside. . . . We, so to speak, cure the patient by preventing him from dying. . . . Herein lies the secret of the treatment of fever. We watch the progress of the disease throughout its varying phases; we meet by judicious treatment, as they arise, the symptoms of secondary and local malady; we sustain the system as far as practicable; we preserve the sufferer at the least expense to the constitution; and we wait patiently until the hour shall strike when in accordance with the mysterious law of periodicity the fever shall have departed and convalescence shall have begun."

The "mysterious law of periodicity" is not now so mysterious as it was in the days of Stokes. The bacterial origin of the malady is everywhere admitted. The symptoms, local and constitutional, are due to a toxæmia. The toxin is resisted by the body-forces, as well as by an antitoxin which the toxæmia calls into being. Then, in the appropriate words of Dr. Andrew H. Smith, "the crisis is deferred until the infecting are balanced by the disinfecting forces, the latter including the various emunctories by which the poison is discharged from the body."

Prophylaxis.—The prevention of pneumonia is a difficult matter. Perfect sanitation of the dwelling-house, civic cleanliness in the fullest sense, the maintenance of a high standard of personal health, and thorough disinfection or destruction of the sputum of pneumonic cases, which is dangerous only in a second degree to that of pulmonary consumption—such are the somewhat Utopian measures by adopting which we may hope to control pneumonia. The subjects of the malady should be isolated and treated in epidemic wards like any other fever patients. The researches of Netter (1888), Foà, and G. and F. Klemperer (1891) suggest the possibility of producing a temporary immunity against pneumonia even in man, by injections of an antipneumococcic serum prepared from an attenuated culture of the *Diplococcus pneumoniæ*. The dog certainly can be rendered immune, and can also be cured of pneumonia by injections of antipneumococcic serum. Man, however, is relatively only slightly susceptible to the pneumonic diplococcus, and therefore we infer that he is equally but slightly susceptible to the antipneumococcic serum.

Management. — A patient suffering from pneumonia should take to bed at the earliest moment in a large, airy, warm, well-lighted room in a well-drained "fresh" house. Pure air and warmth and daylight are magical remedies in this as in other acute diseases. If the house in which the patient resides is badly drained he should at once be removed from its tainted air. Nothing is more striking than the marked improvement which often follows the removal of a pneumonia patient from insanitary surroundings to an airy, well-warmed, wholesome ward in a well-appointed general or private hospital.

Next to the place in which pneumonia is treated comes the manner in which it is nursed. As Graves said years ago, "It is of the utmost importance to economise the patient's strength in fever. The very act of lifting him up, or removing him from one side to another, tends to produce exhaustion." Neither in pneumonia nor in typhus should the patient be allowed to assume an upright position lest cardiac failure should ensue. Hence the necessity for skilled nursing, which will save the sufferer from fatigue in a thousand and one ways. But the nurse must be one such as Shakespeare describes in *Cymbeline*—

> So kind, so duteous, diligent,
> So tender over his occasions, true,
> So feat, so nurse-like.

The skilled nursing of pneumonia is then of paramount importance both by night and by day. It should be unceasing. One care the nurse should have is to prevent the patient talking—in pneumonia, speaking inflicts a far greater strain upon the patient's breathing than in other disease. It also induces fits of coughing, which exhaust the strength.

The dieting of the patient is all-important. As in enteric fever, the food should be liquid, very nourishing, easily assimilable, given in moderate quantities at rather short intervals. More risk results to the patient from over-feeding than from starving. During the fever the

total quantity of liquid food given in 24 hours should not exceed three pints and a half, that is, two litres. In addition, cold water may be allowed *ad libitum*. Should the stomach be much upset and irritable, equal parts of whey and egg-water will usually be borne better than anything else. Egg-water—the *eau albumineuse* of the French—is prepared by whipping up the whites of from two to four eggs to a froth, then stirring into a pint of cold water and finally straining. This albumin-water replaces the casein of the milk which has been separated as curd in the making of the whey. Should a mild stimulant be required, wine-whey may be substituted for ordinary two-milk whey —that is, whey made by adding one part of fresh butter-milk to two parts of warm milk in a saucepan over a slow fire. Milk and soda water, or carbonised water, form a grateful beverage, or the milk may be peptonised or given as "junket." Egg-flip is a good food and stimulant combined. A light and palatable form is made by whipping up the yolk of a fresh egg, sprinkling a little powdered white sugar upon it, adding from a teaspoonful to a tablespoonful of whisky or brandy, and finally pumping soda water from a syphon upon the mixture in a tumbler.

As regards animal broths, it is to be remembered that beef tea as usually made is little more than a stimulant. It is better mixed with chicken broth. Strained mutton broth is very wholesome and palatable. But raw meat juice is much more nourishing than any of these, and mixed with port wine is a powerful restorative.

Freshly made tea with milk or cream may be allowed, and a little toast, if desired, may be soaked in the tea. A cup of black coffee, with or without brandy, is an excellent stimulant in the early morning after a restless night of tossing, sleeplessness, and delirium. Various fresh fruits, such as oranges, grapes, and bananas, may be given throughout the attack. Bananas, indeed, are very nourishing and digestible, and serve as food even in the presence of high fever.

The patient should not be allowed to suffer from thirst. Sipping cold water not only allays the thirst of fever better than anything else, but acts as a stimulant to both heart and liver, as pointed out by Sir Lauder Brunton. Sometimes thirst is relieved by simply holding a little cold or tepid water in the mouth and then spueing it out. Ice is often craved, and should be allowed.

At this time of day it would be idle waste of time to give a detailed historical account of the various methods of treatment adopted in succession for the "cure" of a periodic, self-limited disease. These methods were the antiphlogistic plan, by which it was sought to quench the fires of "*inflammation* of the lungs" by bleeding, calomel, antimony, and opium; the stimulant plan so warmly advocated and energetically practised by Dr. Robert Todd, of London; the wise, if so-called expectant, method adopted by Dr. Hughes Bennet, of Edinburgh, in 1848, which, though somewhat reactionary, recognised the principle of a "vis medicatrix naturæ" in relation to pneumonia; the antipyretic plan of the German school, with its large doses of quinine (30 grains at intervals of 48 hours), its repeated baths at 102° to 104° F., and its ice-pack or cold compresses to the chest walls; the symptomatic treatment of cough, pain, insomnia, delirium, vomiting, and diarrhœa, or other troublesome epiphenomena; and, lastly, the serum or antitoxin treatment, introduced by G. and F. Klemperer in 1891.

Our position to-day is this. We cull from one and all of the foregoing methods of treatment those measures and remedies which appear most likely to benefit our individual patients, without slavishly following one method of treatment to the exclusion of the rest. Let us deal with the subject under two headings: (1) the Antitoxin treatment, (2) Rational treatment.

Antitoxin Treatment.—In their original communication on the nature of the infection in acute fibrinous pneumonia published in 1891, G. and F. Klemperer explain that the crisis seen in pneumonia in human beings occurs at the moment when the poisonous products, manufactured by the bacteria located in the lungs, are present in the circulation in amounts sufficient to call forth in the tissues the reactive change that results in the production of the antidotal substance which has the power of rendering the poisons inert.

At the time of the crisis in pneumonia the bacteria themselves are in no way affected. They remain in the lungs, and can be detected in full vigour and virulence in the sputum of patients a long time after the disease is cured. They have lost none of their power of producing poisonous products, and still possess their original pathogenic relations toward susceptible animals. It is only after the crisis that their poisons are neutralised by this antidotal proteid which has been eliminated by the cells of the tissues, and as this occurs the systemic manifestations gradually disappear. Klemperer and Klemperer isolated, from the cultures of the diplococcus of pneumonia, a proteid body which is the agent concerned in producing the tissue-changes that result in the formation of the protecting substance. They likewise isolated from the serum of immunified animals a proteid which possesses the same powers as the serum itself—of affording immunity and of curing the disease. The poisonous bacterial product they propose to call pneumotoxin; the protecting body, antipneumotoxin.

After obtaining these results upon the lower animals they directed their attention to human beings, and found that by the subcutaneous

application of the serum of immunified animals to patients suffering from acute fibrinous pneumonia the results were in the main promising. They found that while healthy individuals and those suffering from other forms of the disease presented no systemic reaction after the injection of the serum, in 6 cases of pneumonia in which the serum was employed there was a remarkable fall of temperature and slowing of the pulse within the first twelve hours after it was injected. In 4 of these cases the temperature fell to normal, but rose again after six hours. In 2 cases it fell to normal, and remained at that point.

Early in 1897 Dr. J. W. Washbourn, physician to the London Fever Hospital, reported 2 cases of pneumonia treated with antipneumococcic serum. The serum was derived from a pony which had been gradually immunised by injections into the subcutaneous tissue of the shoulder of broth cultivations of the *Diplococcus pneumoniæ* heated to 60° C. for one hour, then of living agar cultivations, and, lastly, of living broth cultivations. In both cases the treatment appeared to exert a beneficial effect upon the disease. Washbourn recommends that the serum should be injected into the subcutaneous tissue with strict aseptic precautions. The dose should be 20 cubic centimetres (660 units). The treatment should be commenced as early as possible in the attack, and should be repeated twice a day until the patient is convalescent. Andrew H. Smith thinks that we must conclude that up to the present the achievements of orrhotherapy in its application to pneumonia can scarcely be said to amount to more than an encouragement to further effort. "No really decisive results have been obtained. In some cases the effect seems to have been favourable, but in view of the variable course of pneumonia under all forms of treatment, it is impossible to assign to the injections any positive share in the result."

Rational Treatment.—Ever keeping before his mind the idea of the specific nature of pneumonic fever, the object of the physician in treating this disease should be to guide the patient safely back to health through the manifold dangers by which he is encompassed.

The bowels should be kept active once or twice a day. The diet should be regulated. Sleep should be secured.

Temperature may run so continuously high (104° F., or upwards) as to suggest a risk of heart failure, apt to be induced by the action of pyrexia, combined with the profound disturbance of the pulmonary circulation caused by the pneumonia. At the same time, we should recollect that the rate of mortality is higher in the presence of relatively low temperatures (under 103° F.) than it is when the thermometer ranges between 104° and 105°. The German doctrine of "Das Heil-fieber" holds good in the case of pneumonia as in that of other continued fevers. Elevation of body temperature means a reaction of the body forces against the toxæmia. Cold water, applied externally and taken internally, is the safest of all antipyretics. It lowers temperature without interfering with the production of body-heat (thermogenesis). Sir Lauder Brunton cites, as the most striking example he ever saw of the use of cold, the case of a patient suffering from pneumonia, who was dying from hyperpyrexia. At the suggestion of a Swedish physician who was present, Professor J. Hughes Bennet, of Edinburgh, under whose care the patient was, applied the wet pack. A big tub of cold water was ordered into the ward. All the bedclothes were pulled off, a sheet was dipped in the water, and the patient was wrapped in it. In a few minutes it was taken off and a second cold sheet was applied. This was repeated again and again, and in an hour the patient lay quiet and comfortable, apparently in an easy slumber. From that time forward the patient went on without a bad symptom, and in due course recovered perfectly.

Sponging with vinegar and tepid water (one part in four) is a useful modification of the bathing treatment of pneumonia.

Another risk to life arises from collateral œdema of the lungs or brain, and this in a sthenic patient with high fever may be relieved by leeching or venesection, remedies which have been too much neglected of late years. Cyanosis, stupor, transitory paralysis affecting one or other side of the body (hemiplegia), rapid respirations, and a serous frothy expectoration, are indications of collateral hyperæmia, which a moderate venesection—4 to 6 or 8 ounces of blood—would probably at once relieve. The writer can call to mind three or four cases of this kind, in which venesection did good, and quickly.

The administration of alcoholic stimulants calls for much circumspection on the part of the physician. Many—probably most—pneumonic patients are better without stimulants, which may tire the heart and congest the liver. The intemperate and the aged require stimulants—the former in moderation, the latter often *pleno rivo*. It is a good plan to combine stimulants and food, as in egg-flip, wine-whey and egg-water, milk and whisky or brandy. For an emergency champagne plays a useful part. Women and children often bear stimulants badly. Under all circumstances, the ordering of these dangerous remedies should be subject to revision day by day.

As secondary, though useful, aids to treatment, several local applications may be mentioned. A single leech applied early in the attack will often relieve the distressing "stitch in the side" of that stage. When the leech falls off the wound may be allowed to bleed into a linseed-meal poultice for a quarter of an

hour or twenty minutes. A relay of hot linseed-meal poultices is an old-fashioned and useful practice. Mustard in varying proportions, when mixed with such a poultice, produces more decided counter-irritation, and prevents the uncomfortable feeling of chill which ensues on cooling of the poultice. In children a jacket of wadding is a good substitute for the poultice. The inflammable nature of wadding should not be forgotten. Turpentine stupes do good, particularly when there is coincident bronchitis. Dry cupping is excellent practice for the relief of pain and dyspnœa, but also as a preventive of collateral hyperæmia. Blistering is often harmful, and when prescribed should be used with moderation. Free and reckless blistering may damage the burdened kidneys, or actually set up a traumatic pleuropneumonia. Mention has already been made of the application of ice-cold cloths to the affected side, and even the ice-bag may be employed with advantage.

Two vital indications in treatment are—to support the heart and keep it going, and to prevent clotting of the blood.

The first indication is met by ordering digitalis or strophanthus, in full doses, singly or in combination with quinine, nux vomica, or strychnine. Digitalis is indicated when the pulse is rapid and of low tension, and when the kidneys do not act freely. This drug is well borne in pneumonia, and may be pushed. By some authors it is credited with an almost specific action on the disease. Strophanthus has the advantage of acting more quickly upon the heart, and may be given in high tension cases. Another useful drug is caffein, given as an effervescent citrate or hydrobromate, with or without 10-grain doses of salicylate of sodium. Caffein should be withheld late in the afternoon and towards night lest wakefulness should result. In threatened heart failure hypodermic injections of strychnine ($\frac{1}{30}$-$\frac{1}{15}$ grain) every eight or twelve hours often save life. Morphine may be given with it ($\frac{1}{8}$-$\frac{1}{6}$ grain) if the patient is restless and sleepless.

Blood-clotting may be anticipated by the exhibition of neutral salts, or of carbonate of ammonium in 5-grain doses every third, fourth, or sixth hour. A pill of 3 grains each of ammonium carbonate and quinine is a suitable form. Aromatic spirit of ammonium serves the same purpose; in urgent cases it may be combined with digitalis, Hoffmann's anodyne (compound spirit of ether), and tincture of bark. Inhalations of oxygen give great relief, and once tried are often craved for by the patient. The mouthpiece of the inhaler should not be taken into the mouth, for the concentrated stream of oxygen dries the tongue and parches the mouth. The writer has never seen any harm from the inhalation of diluted oxygen, the nozzle being held a couple of inches from and below the open mouth.

For the relief of cough the local applications already mentioned should be tried. Sipping cold water gives relief, or half an ounce of both glycerine and lemon juice may be put into a 2-ounce bottle, which should then be filled with water; of this simple mixture a teaspoonful may be sipped occasionally. To such a mixture cherry-laurel water, or syrup of codein, or solution of morphine, might be added to produce a more decided sedative effect. Sir William Jenner's cough mixture for phthisis should do good in some cases, but it is apt to check expectoration, namely:

℞ Acidi hydrocyani diluti, ♏xlviij.
Liquoris morphinæ acetatis, ♏xcvj.
Syrupi aurantii, ad ℥iij.
Signa: A teaspoonful, by measure, to be sipped occasionally.

For the early headache and sleeplessness, the phenazone and gelsemium draught recommended in the article on "Influenza" may be prescribed. The formula for this magical remedy is as follows:—

℞ Phenazoni, gr. xij.-xx.
Tincturæ gelsemii, ♏xx.
Aquæ chloroformi, ad ℥ij.
Signa: One-fourth part every second or third hour until pain is eased.

As a rule, narcotics, like opium or morphine, had best be avoided in the treatment of pneumonia. They lock up the secretions and check expectoration. Perhaps an exception may be made in favour of Dover's powder, which is useful when given in a pill with quinine and digitalis (Niemeyer's modification of Heim's pill).

The bromides are suitable hypnotics, exhibited with tincture of bark. If the heart is strong, and early in the attack, chloral may be given in combination with the bromides of sodium, potassium, and ammonium; later on chloral is inadmissible except in young children, who bear the syrup well. A teaspoonful may be prescribed with 7 drachms of simple syrup or of aromatic syrup, the doze being one or two teaspoonfuls of this mixture at suitable intervals.

Chloralamide and bromide of potassium, of each 15 to 30 grains, sometimes act well in soothing and so inducing sleep. They may be dispensed flavoured with liquid extract of liquorice, or with tincture of orange peel and chloroform water.

Delirium is sometimes active in apical pneumonia, but it is seldom persistent. It is allayed by whatever makes the patient less restless and more comfortable. Sipping cold water for the relief of thirst, the application of cold compresses to the affected side, the relief of tympanites or flatulence or constipation, the application of warmth or cold to the fore-

head, the use of the menthol cone or of ice in stroking the brow—these simple means should in the first instance be tried. Only when they fail should more active measures be resorted to.

In coincident bronchial catarrh, or where "purulent infiltration" of the lung is taking place, turpentine is a sheet-anchor. It may be given in the form of capsules, or in punch or mixture. Turpentine punch is best made by rubbing a little lemon rind on a lump of white sugar, then dropping 15 to 20 minims of spirit of turpentine upon the sugar, and dissolving it in a wine-glassful of whisky punch.

Turpentine is prescribed in a mixture as follows:—

℞ Spiritus terebinthinæ, ʒiij.
Ovi vitellum unum.
Tere et misce bene—
Spiritus ætheris nitrosi, ʒiij.
Spiritus chloroformi, ʒij.
Aquæ menthæ piperitæ, ad ℥viij.
Signa: Half an ounce to an ounce for a dose.

Other complications must, of course, be dealt with as they arise, but on all occasions and in all circumstances the physician should remember to avoid the "nimia diligentia medici" in treating a disease like pneumonia, which so pre-eminently is periodic, and tends to get well of itself, perhaps in spite of a fussy and ill-named "heroic" treatment.

Pneumonitis.—Pneumonia (*q.v.*)

Pneumono-. — In compound words *pneumono-* (Gr. πνεύμων, the lungs) means relating to the lungs, pulmonary.

Pneumonocele.—Hernia of the lung.

Pneumonokoniosis.—This term (Gr. πνεύμων, lung, and κόνις, dust) is given to all the affections of the lungs in which there is chronic inflammatory induration of the pulmonary tissues due to the irritation produced by inhaled particles of coal dust (*anthracosis*), of metals such as iron or steel (*siderosis*), or of minerals such as silica (*chalicosis*). *See* Lungs, Pneumonokoniosis; Lung, Tuberculosis (*Predisposing Factors*).

Pneumonomycosis.—Disease of the lungs due to fungi. *See* Lungs, Parasitic Affections of (*Aspergillosis*, etc.).

Pneumopericardium.—The presence of air or gas in the pericardial sac. *See* Pericardium, Diseases of (*Pneumopericardium*).

Pneumoperitoneum.—The presence of air or gas in the peritoneal cavity.

Pneumopyothorax. — The presence of air and pus in the pleural sac.

Pneumorrhagia. — Pulmonary apoplexy or hæmorrhagic infarct.

Pneumothorax.—The presence of air or gas in the pleural cavity. *See* Aspiration (*For Therapeutic Purposes*); Mediastinum (*Mediastinal Emphysema*); Peritoneum, Acute Peritonitis (*Subphrenic Abscess, Diagnosis*); Pleura, Diseases of (*Pneumothorax*); Pleura, Surgical Affections of (*Injuries causing Pneumothorax*); Typhoid Fever (*Complications*).

Pneumotomy.—Incision of the lung.

Pneumotoxine.—The poisonous albuminoid substance produced by the pneumococcus and believed to be the cause of many of the symptoms of pneumonia; an antitoxin (*antipneumotoxine*) is believed to be present in the blood of the patients who recover.

Pneumotyphus or Pneumotyphoid. — Pneumonia occurring during typhoid fever or pneumonia due to the typhoid bacillus. *See* Typhoid Fever (*Symptoms*).

Pock.—A vesicle or pustule such as forms in smallpox or in chickenpox; also, the disease in which such an eruption appears, *e.g.* the great pox or syphilis and the smallpox or variola.

Podagra.—Gout, especially the typical form of the disease in which the great toe is affected (Gr. πούς, foot, ἄγρα, a catching). *See* Gout.

Podalgia.—Pain in the foot, as in gout.

Podalic Version.—That form of turning the fœtus during labour in which the feet are brought out first.

Podencephalus.—That form of monstrosity in which the upper part of the cranium is defective and allows the contents to lie exposed; median mero-acrania.

Podex. — The rump or gluteal regions (Lat. *podex*, the anus or fundament).

Pododynia.—Pain in the foot, especially when neuralgic in kind.

Podophylli Resina.—Obtained from the dried rhizome of *Podophyllum peltatum*, the American mandrake or May apple. It is also known as *podophyllin*. *Dose*—$\frac{1}{4}$-1 gr. *Preparation*—Tinctura Podophylli. *Dose*—5-15 m. A non-official *Tinctura Podophylli Ammoniata* is also prepared, *dose*—5-20 m., which has the advantage that the addition of water to it does not precipitate the resin.

Podophyllin is used only for its purgative action. In small doses it is laxative, in large ones cholagogue. It is very slow in its action, and, if given in combination with more rapidly

acting purgatives, it may be swept away before any effect has been produced. It is especially useful in biliousness, sluggish hepatic action, and hepatic cirrhosis. It causes considerable griping, and should therefore be combined with hyoscyamus or some other drug which will prevent this. It forms an important constituent of many "dinner pills." In some cases of summer diarrhœa in children and of chronic diarrhœa in adults minute doses of podophyllin frequently repeated have a beneficial effect.

Podophyllum, Indian. — In the Indian and Colonial Addendum to the British Pharmacopœia, the dried rhizome and roots of *Podophyllum emodi* (under the name of *Podophylli Indici Rhizoma*) as well as the resin obtained therefrom (*Podophylli Indici Resina*) are officinal. From the latter the *Tinctura Podophylli Indici* (dose, 5 to 15 m.) is prepared. Indian podophyllum is used for the same purposes as the podophyllum of the B.P.

Poggi's Corpuscles. — Red blood corpuscles, probably in an immature state, which "stain with basic dyes in the fresh, unfixed specimen" (*Da Costa*).

Pogoniasis.—The excessive growth of the beard in a man, or the development of hair on the chin of a woman (Gr. πώγων, a beard).

Poikilo-. — In compound words *poikilo-* (Gr. ποικίλος, variegated) means irregularly shaped; thus *poikilocytosis* means that condition of the blood in which it contains *poikilocytes* or deformed red blood corpuscles (most often the deformity is crenation, but other irregularities are met with). *See* Chlorosis (*Pathology, Poikilocytosis*).

Point. — A defined place or spot. *See* M'Burney's Point.

Poisoning. *See* Toxicology; Pyæmia; Septicæmia; etc.

Poison Nut.—A synonym of nux vomica (*q.v.*).

Poisons. — *See* Catheters, Uses and Dangers (*Septic Poisoning*); Food (*Effects of eating Impure Food*); Medicine, Forensic (*Post-mortem Examination in Cases of Suspected Poisoning*); Morphinomania; Pregnancy, Intra-Uterine Diseases (*Effect of Poisons on the Fœtus and Placenta*); Snake-Bites and Poisonous Fishes (*Poisonous Fish*); Toxicology; Trades, Dangerous (*Lead-Poisoning*, etc.).

"Poker-Back."—Rigidity of the spine with anchylosis of the vertebræ due to arthritis deformans or other disease; spondylitis deformans.

Polar Bodies.—The small globules or bodies thrown off by the ovum during the process of maturation. *See* Fœtus and Ovum, Development of (*Ripening of the Ovum*).

Pole.—Each of the extremities of the axis of a body, either spherical or spheroidal or ovoid, *e.g.* the crystalline lens of the eye, the fœtus in utero; in electricity each of the terminal points of a battery is termed a pole (positive and negative).

Polenta. *See* Pellagra (*Causes, Maize Polenta*).

Policlinic.—Medical instruction given in the houses of patients in a town, or, more recently, in a dispensary or out-patient department of a hospital (Gr. πόλις, a city, κλίνη, a bed).

Poliencephalitis. — Inflammation of the grey matter of the brain (Gr. πολιός, grey, ἐγκέφαλος, the brain).

Polio-.—In compound words *polio-* (Gr. πολιός, grey or hoary) means grey.

Polioencephalomyelitis.—Inflammation of the grey matter of the brain and spinal cord. *See* Myasthenia Gravis (*Diagnosis*).

Poliomyelitis. — Inflammation of the grey matter of the spinal cord. *See* Paralysis (*With Atrophy of Muscles, Acute and Subacute Anterior Poliomyelitis*). *See also* Deformities (*From Cerebral and Spinal Paralysis*).

Polioplasm.—Granular protoplasm.

Poliosis or Poliothrix.—Canities; greyness, premature or senile, of the hair. *See* Canities and *Cross References.*

Politzer's Bag.—An india-rubber bag used for inflating the middle ear, inflation so produced being termed *Politzer's Method* or politzerisation. *See* Ear, Examination of (*Inflation through the Eustachian Tube*).

Pollakiuria. — Abnormal frequency of micturition (Gr. πολλάκις, many times).

Pollantin.—A powder recommended for the treatment of hay fever; it is applied directly to the nostrils or conjunctiva; it is also used in a liquid form; and it is said to give relief in a certain number of cases.

Pollen.—*See* Hay Fever (*Etiology*).

Pollex.—The thumb; thus, many of the muscles of the hand have names in which *pollicis* (of the thumb) is included, *e.g. flexor brevis pollicis*, etc.

Pollution. *See* Water (*Sources of Impurity*).

Poltfoot.—Club-foot. *See* Deformities.

Poly-.—In compound words *poly-* (Gr. πολύς, much, and πολλοι, many) means many.

Polyadenia. — Hodgkin's disease, or pseudoleukæmia.

Polyadenomata.—Polypi, *e.g.* of the stomach.

Polyadenopathy.—The disease of several lymphatic glands at the same time.

Polyæmia.—An increase in the volume of the blood, unaccompanied by any change in its composition; plethora. *See* Leucocythæmia (*Changes in the Blood*).

Polyæsthesia.—A rare morbid condition of the skin in which sensation is so disturbed that the point of a pin is felt as two or more points.

Polyarthritis.— The affection of several joints at once, as in rheumatism, which is then called *polyarticular*. *See* Rheumatism, Rheumatoid Arthritis (*Synonyms*).

Polyaxon.—A neuron with more than two axons.

Polyblast.—The morula mass or mass of many cells due to the segmentation of the ovum. *See* Fœtus and Ovum, Development of.

Polyblepharon.—The presence of a supernumerary eyelid; the membrana nictitans.

Polycholia.—Excessive secretion of bile. *See* Jaundice (*Pathology, Jaundice from Polycholia*).

Polychromatophilia. — A morbid condition of the blood in which "some of the corpuscles lose their affinity for the acid stain, and with mixtures of both acid and basic dyes are stained atypically by either or both elements" (*Da Costa*).

Polychromia.—The increased formation of pigment, *e.g.* of the bile. *See* Jaundice (*Pathology, Hæmatogenous Jaundice*).

Polyclinic.—This term is sometimes used as if it were synonymous with *policlinic*; but its meaning is, strictly speaking, an institution in which all kinds of diseases are gathered together for the purposes of treatment and of providing clinical instruction; the name has also been given, although the use is hardly accurate, to an institution in which there are none but out-patients.

Polycoria.—The presence of more than one pupil in the eye. *See* Iris and Ciliary Bodies (*Congenital Abnormalities*).

Polycrotic Pulse. — A pulse the sphygmogram of which shows several crests in each wave. *See* Physiology, Circulation (*Characters of Pulse Wave*).

Polycyesis.—Multiple or twin pregnancy, or, less often, frequent child-bearing.

Polycystic.—Containing several cysts, *e.g.* an ovarian tumour with several separate cavities.

Polycythæmia.—A morbid state of the blood in which the number of red blood corpuscles (*erythrocytes*) is increased.

Polydactylism.—The malformation in which more than the usual number of digits is present. *See* Deformities (*Hand and Fingers*); Heredity (*Inheritance of Polydactylism*); Mental Deficiency (*Diagnosis*).

Polydipsia.—Excessive thirst.

Polygalactia.—Excessive secretion of milk. *See* Infant Feeding (*Human Milk, Variations in Quantity*).

Polygalic Acid.—A glucoside found in senega root (*Polygala Senega*).

Polygenism.—The theory that mankind arose from several independent pairs of ancestors. *See* Anthropology.

Polygnathus.—A form of double monstrosity in which the parasitic fœtus is attached to the jaw of the autosite.

Polygraph. *See* Pulse (*Pulse and Respiration, Clinical Polygraph*).

Polygyria.—The condition of the brain in which the number of cerebral convolutions is increased.

Polyhæmia. *See* Polyæmia.

Polyhydramnios.—Excess of liquor amnii in pregnancy. *See* Labour, Faults in the Passenger (*Excess of liquor amnii*); Pregnancy, Pathology of (*Affections of Amnion*).

Polylobular.—Having many lobes, *e.g.* some fibroid tumours.

Polymastia.—The presence of more than two mammæ in the human subject. *See* Mammary Gland, Diseases of (*Congenital Anomalies*); Teratology (*Malformations of Thorax*).

Polymelia.—The presence of more than the normal number of limbs, *e.g.* an accessory limb attached to the thoracic or pelvic girdle.

Polymerisation.—*See* Physiology, Food and Digestion (*Carbohydrates, Polymerisation of Monosaccharids*).

Polymorphic.—Having several forms,

e.g. the polymorphic coccus of Cedercrentz. *See* SKIN, BACTERIOLOGY OF.

Polymorphs.—Leucocytes having variously shaped nuclei consisting of apparently separate portions; polymorphonuclear leucocytes.

Polymyositis. — An inflammatory disease of the muscles. *See* MUSCLES, DISEASES OF (*Inflammatory*).

Polyneuritis. *See* NERVES, PERIPHERAL (*Multiple Peripheral Neuritis*); PARALYSIS (*Landry's Paralysis, Diagnosis*).

Polynuclear.—Having several nuclei; multinuclear.

Polyœstrous. — Having a series of cycles of reproductive activity (*œstrus*) in the same year, *e.g.* sheep and mares.

Polyopia or Polyopsia.—Multiple vision, or that morbid state of the eyes in which one object is seen as two or more. *See* CATARACT (*Senile, Diagnosis*).

Polyorchidy or Polyorchism.—The rare abnormality in which more than two testicles are present. *See* SCROTUM AND TESTICLES, DISEASES OF (*Abnormalities*).

Polyorrhomenitis or Polyorromenitis.—Suppuration or tuberculosis affecting several serous membranes; Concato's Disease. *See* PERITONEUM, TUBERCULOUS PERITONITIS (*Varieties*).

Polyotia. *See* EAR, EXTERNAL, DISEASES OF (*Malformations of Excess, Cervical Auricles*).

Polyparesis. — Paresis affecting many muscles; general paresis or paralysis.

Polypeptides. *See* PHYSIOLOGY, PROTOPLASM (*Synthesis of the Products of Disintegration*).

Polyphagia. — Excessive appetite for food; bulimia. *See* APPETITE (*Increase of*).

Polypharmacy.—The use of many drugs in the treatment of disease.

Polyphrasia.—Verbosity, especially in insanity; logorrhœa.

Polyphyodont.—That form of dentition in which there is a continuous succession of teeth throughout life; it is not found as high as the mammalia, mammals and man being *diphyodont* (having two sets).

Polypnœa.—Rapid or panting respiration.

Polyposia. — A passionate desire for drinking; dipsomania.

Polypotome or Polyptome.—An instrument for removing a polypus (*q.v.*).

Polypus. — A rounded, pear-shaped, or branched tumour, usually provided with a pedicle, springing from a mucous or serous surface. *See* EAR, MIDDLE, CHRONIC SUPPURATION (*Complications, Polypi*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Morbid Process, Thrombosis in Heart*); MOLE, MOLAR PREGNANCY (*Placental Polypus*); NOSE, DISEASES OF NASAL ORIFICES AND SEPTUM (*Benign Tumours of Nasal Fossæ*); NOSE, ACCESSORY SINUSES, INFLAMMATION OF (*Pathology*); RECTUM, DISEASES OF (*Polypus Recti*); UMBILICUS, DISEASES OF (*Tumours*); UTERUS, NON-MALIGNANT TUMOURS OF (*Polypi*).

Polysaccharid. *See* PHYSIOLOGY, FOOD AND DIGESTION (*Carbohydrates, Polysaccharids*).

Polysarcia.—Obesity (*q.v.*)

Polyserositis. *See* POLYORRHOMENITIS.

Polysomia.—The teratological state in which two fœtuses (complete or imperfect) are united together; fused twins.

Polyspermy. — The entrance of more than one spermatozoon into the ovum in impregnation; it is one of the alleged causes of double monsters.

Polystichia.—The arrangement of the eyelashes in two or more rows.

Polythelia.—The presence of more than two nipples (one on each mamma) in the human subject; the accessory nipple may be on the chest, in the axilla, or on the abdomen. *See* MAMMARY GLAND, DISEASES OF (*Congenital Anomalies, Polymastia*).

Polytocous. — Producing numerous offspring, or prolific.

Polytrichia.—Excessive hairiness. *See* HIRSUTIES.

Polyuria.—The excessive secretion and excretion of urine. *See* BRAIN, TUMOURS OF (*Localising Symptoms, Urine*); HYSTERIA (*Organs of Secretion, Kidneys*); KIDNEY, SURGICAL AFFECTIONS OF (*Movable Kidney, Symptoms*); KIDNEY, SURGICAL AFFECTIONS OF (*Traumatic Nephritis, Symptoms*); KIDNEY, SURGICAL AFFECTIONS OF (*Renal Tuberculosis, Symptoms*); MICTURITION (*Frequency*); OCULAR MUSCLES, AFFECTIONS OF (*Paralysis, Etiology, Position of Lesion*); URINE, PATHOLOGICAL CHANGES IN (*Variations in Quantity*).

Pomegranate Bark. *See* GRANATI CORTEX.

Pompholyx.—A vesicle on the skin, or a

cutaneous disease characterised by the eruption of vesicles, fever being absent (Gr. πομφόλυξ, a bubble).

Pomum Adami. — Adam's apple, the fanciful name given to the projecting angle formed by the meeting of the two alæ of the thyroid cartilage of the larynx in the neck.

Ponfick's Corpuscles or Shadows.—Colourless blood corpuscles or "phantoms" produced by morbid changes in the erythrocytes; Hayem's achromacytes.

Ponograph.—An instrument for estimating and registering sensitiveness to pain (Gr. πόνος, pain).

Ponos (πόνος, suffering, pain) is a peculiar endemic disease, affecting young children only, occurring in the Greek islands of Hydra and Spezia off the coast of Argolis. The disease is characterised by marasmus, enlargement of the spleen (usually), and a characteristic odour of the urine. The mortality is very great, either from the marasmus, hæmorrhage, or general collapse. Röser first directed attention to this disease in 1835, and Pallas called attention to the hæmorrhagic phenomena and their resemblance to those of scurvy in 1842, but it was not until 1871 that the disease was generally recognised by Grecian physicians. It is probably not so frequently met with now as it once was. Its origin is unknown. Jeannopoulos thought it was due to malarial infection, but this is not so. Climate and season have no influence upon its production, nor has the constitution of the soil. It is met with in rich and poor alike, and food and water seem to have no effect in its production. But it seems to be limited to certain families, and is popularly supposed to be due to hereditary conditions, or at any rate to congenital predisposition, for the parents of children suffering from this disease are usually found to have suffered from some serious disease, especially tuberculosis. Karamitsas held that the disease was a kind of splenic leukæmia, but this view is not supported by either the symptoms during life or the post-mortem appearances. Hirsch describes it as a constitutional affection deeply rooted in errors of nutrition. Scheube considers that the disease much resembles the liver cirrhosis met with in India. In all probability some parasite will eventually be found to be its cause. The duration of the disease varies from two or three months to one or two years. It is rarely seen in children over four years of age, and Stephanos considers it rather more common in boys than in girls. Of the pathology of the disease we are quite ignorant.

The symptoms of ponos vary. The child suffers from fever of an intermittent or irregular type, fluctuations from first to last; if it assumes the character of hectic fever, death nearly always results; the child becomes feeble, pale, emaciated, disinclined to play, but the appetite is usually good; it may indeed become ravenous. *Pari passu* the spleen enlarges and is painful as a rule, and it is this pain which gives the name to the disease, but in some cases it is entirely absent, although the spleen is greatly enlarged. Constipation is the rule at first, followed later on by diarrhœa, sometimes severe. From the first and throughout the disease the urine has a peculiar characteristic penetrating odour, and bronchial catarrh usually obtains throughout the whole course of the disease. After the onset of diarrhœa, œdema occurs, followed by ascites and sweating; and then hæmorrhage from the nose, bowels, and especially from the gums, occurs, together with ecchymoses of the skin. Pneumonia, meningitis, and peritonitis are apt to complicate matters, and lead to a speedily fatal termination of the disease.

Treatment.—This is unsatisfactory. If the child is attacked when very young a good wet-nurse should be procured. Good food and tonics, such as iodide of iron, quinine, iron, are indicated; change of air should be advised, and, on the bare chance that the disease may be possibly combined with a scorbutic element, plenty of ripe fruit should be given.

Dr. George I. Williamson, of Larnaca, Cyprus, writing of ponos, says: "I have for some considerable time been on the outlook here for cases that might be so classified, but have never met with any. Many cases of malarial fever in infants seem to closely resemble the disease as described, say, in Manson's *Tropical Diseases*. From inquiries made, I find that ponos has never been even suspected to exist in Cyprus. Dr. Trepis, of Larnaca, a native of Hydra, writes me that the older medical men of that island considered ponos to be tubercle of the mesenteric glands and peritoneum, and that the inhabitants thought it contagious and infectious, as many children of the same family were frequently attacked; he adds that he does not know how the disease is now considered by the doctors of Greece, but his own idea is that it is not a disease *per se*."

I have lately come across an article on ponos written by Dr. Jeannopoulos, who practised in Spetsee for a considerable number of years: his article appeared in a Greek medical journal in 1879, and so is of earlier date than the descriptions by Karahistsa (1880) and Stephanos (1881). Jeannopoulos, whose article I have lately translated from the original Greek, considers the most suitable name to be septic marsh fever (ἑλογενὴς πυρετὸς σηπεδονήδης), the title ponos (which he considers given by reason of the gangrene frequently noted) appearing unsuitable, as the terms πόνος and πονερά are used throughout Greece for any sore or wound. His idea as to etiology is that a marsh miasm

from the large marsh Berberonta is carried by the wind to Hydra and Spetsee, the sea being a good conductor of this miasm, which is, however, impeded by the presence of hills.

He mentions as symptoms the following—Beginning as an ordinary intermittent fever, it later becomes irregular; loss of strength and appetite, swelling of abdomen, characteristic septic odour of urine, straw colour of face and body; enlargement of spleen, but not of liver; necrosis of the jaws, especially the upper. Duration—months, not weeks.

Microscopically—no increase of leucocytes.

Boys and girls attacked equally often.

Diagnosis.—From dentition, leukæmia, malarial fever.

Treatment.—Quinine, good food, nursing, wine, cauterisation of gangrenous patches.

Pons. *See also* Auditory Nerve and Labyrinth (*Localisation of Lesion in Nerve-Deafness*); Brain, Physiology of (*Blood-Supply of Pons*); Brain, Affections of Blood-Vessels (*Vascular Lesions of Pons*); Brain, Tumours of (*Pons*).—Lesions of the pons are of interest to the practitioner in at least two directions. Firstly, in cases where there is a gross lesion of the pons of a nature demonstrable to the naked eye, *e.g.* tumour growth; and in the second place there is a wider group of so-called nervous cases where some of the symptoms present are directly traceable to disturbance in this part of the central nervous system. For the due appreciation of either case we must have a clear conception of the physiology of the part, noting specially—

1. The part which the pons plays as the transmitter of motor impulses from the brain cortex.

2. Its function as a transmitter of sensory impressions from the periphery.

3. The function of its grey matter as the nuclei of origin of the cranial nerves, especially the 5th, 6th, and 7th, and the close relationship of the 6th nucleus to the nucleus of the 3rd nerve, whereby a lesion of the nucleus of the 6th nerve is attended by paresis or paralysis of that nerve, and also paresis of the internal rectus muscle on the other side (Conjugate Paralysis).

Bearing these in mind we will readily understand how a localised lesion in this region may produce very definite symptoms of a unilateral or bilateral nature; and if we bear in mind that the fibres of the facial nerve decussate in the lower half of the pons, at a level lower than the crossing of the pyramidal tract, we will understand how a condition of alternate or crossed paralysis (*vide* vol. i. p. 496) may be brought about.

The diagnosis of a localised lesion in the pons is as a rule fairly easy, and is made from a study of the positive and negative symptoms present in each case (*see* art. on "Brain, Tumours of," vol. i. p. 486). It is much more difficult, and frequently impossible in cases of a more general nature, to determine how far the various motor or sensory disturbances are due to lesions in the pons rather than to disease in a region above or below that level. And, fortunately, in most cases the differential diagnosis is immaterial. The cause of the difficulty lies in the tendency for the occurrence of widespread vascular disease in these cases. Some diseases, for long regarded essentially lesions of the nervous system, are now looked upon as more of the nature of a general toxæmia with the chief local incidences in the nervous system, where the toxic agencies at work have exerted their influences primarily on the blood-vessels. In view of this, in all so-called nervous cases, more especially those of an ill-defined nature, it is well to carefully consider how far the anomalous symptoms may be due to vascular lesions in the pons or other part of the central or peripheral nervous system. This can only be determined by a careful study of the history and condition of each case, and in some cases the diagnosis of such vascular lesions can only be arrived at by a process of exclusion. The lesions of the pons met with in cases of cerebral hæmorrhage and in cases of bulbar paralysis are referred to in their special sections (vol. i. p. 458, and vol. vii. p. 300).

Ponticulus. — A small ridge on the cranial aspect of the cartilage of the external ear.

Pontresina. *See* Therapeutics, Health Resorts (*Switzerland*).

Popliteal.—Belonging to the hollow at the back of the knee (Lat. *poples*, the ham); the muscle in this region, therefore, is called the *popliteus*. *See* Aneurysm (*Popliteal*); Arteries, Ligature of (*Popliteal*); Knee-Joint, Diseases of (*Bursæ in the Popliteal Space*); Lymphatic System (*Glands*); Nerves, Peripheral (*Diseases of Special Spinal Nerves*); Syphilis (*Tertiary, Vascular System, Popliteal Artery*).

Poppy. *See* Opium.

Population. *See* Vital Statistics (*Population*).

Porcupine Disease. *See* Ichthyosis (*Ichthyosis Hystrix*).

Porencephalus.—A state of defective development (congenital or acquired) of the cerebrum, consisting in the presence or formation of small spaces or depressions, some of which may open into the ventricles. *See* Athetosis (*Morbid Anatomy*); Mental Deficiency (*Congenital Cases, Porencephalic*).

Poretta. *See* Balneology (*Italy, Sulphur Waters*).

Porocephalus Constrictus. *See* Parasites (*Arthropods, Arachnoidea*).

Poroplastic.—The name given to a kind of porous felt which becomes plastic when heated and can therefore be moulded to the body; it is used as a splint in various surgical conditions.

Porrigo.—The name given somewhat loosely to various skin diseases characterised by scaly eruptions (Lat. *porrigo*, scurf), *e.g. porrigo favosa* or favus. *See* Impetigo.

Porro's Operation. *See* Labour, Obstetric Operations (*Cæsarean Section, Porro's Operation*); Pregnancy, Affections of Generative Organs (*Fibroid Tumours and Pregnancy*); Pregnancy, Hæmorrhage (*Accidental Hæmorrhage, Treatment*).

Porta.—The hilus or "gate" of an organ, through which the vessels and other structures enter the organ, *e.g.* the *porta hepatis*, while the vein entering the porta hepatis is called the *portal vein* or *vena portæ*.

Portal Vein. *See* Ascites (*Causation, Portal Cirrhosis*, etc.); Liver (*Portal Thrombosis*).

Porte-Caustique.—An instrument for applying caustic (*e.g.* nitrate of silver) to a part of the body, *e.g.* the corpus or cervix uteri.

Porter-Clark's Process.—A method of removing hardness from water by adding hydrated calcium oxide and removing the precipitate by filtration through canvas under pressure.

Portio.—A part, *e.g.* the cervix uteri, and *portio dura*, the facial nerve (the auditory nerve being the *portio mollis*).

Portobello. *See* Therapeutics, Health Resorts (*Scotch*).

Portugal. *See* Balneology (*Spain and Portugal*).

Port-wine Mark. *See* Capillaries, Diseases of (*Nævus*).

Porus.—A small opening or pore, *e.g.* the *porus opticus*, through which the central artery of the retina passes.

Position.—The posture or attitude into which a patient is put for purposes of examination or operation (*e.g.* the *genu-pectoral* position, the *lithotomy* position, *Sims'* position, etc.); or the relation which the fœtus in utero bears to the pelvis of the mother (*e.g.* the left occipito-anterior position, the right mento-posterior position, etc.). *See* Labour, Diagnosis and Mechanism (*Positions*).

Posology.—The department of medicine which has to do with the dosage of drugs. *See* Prescribing; Metric System; and under the various drugs.

Possession.—The mental state associated with wildness or extravagance of behaviour or with actual insanity; the name originated in the old belief that a person so affected was possessed by a demon or evil spirit. *See* Insanity, Nature and Symptoms; Lunacy; Medicine, History of; etc.

Post-. — In compound words *post-* (Lat. *post*, after or behind) means after (in time) or behind (in space), *e.g. post-choreic* (following chorea), *post-embryonic* (after the embryonic stage is past), *postanal* (behind the anus), *post-ethmoid* (behind the ethmoid bone), and many other words.

Postapoplectic Insanity.—Insanity following upon an apoplectic seizure. *See* Insanity, Nature and Symptoms (*Etiological Varieties*).

Postcentral.—Situated behind the central fissure (fissure of Rolando) of the brain, *e.g.* the *sulcus postcentralis inferior*, the *postcentral line*, etc.

Postclimacteric.—Occurring after the climacteric or menopause, *e.g. postclimacteric metrorrhagia, postclimacteric insanity*, etc.

Postconnubial.—Occurring after marriage, *e.g.* postconnubial insanity; postnuptial.

Postcubital.—Situated behind the forearm.

Postdiastolic.—Following the dilatation or diastole of the heart.

Postdicrotic. — Occurring after the dicrotic wavelet of the sphygmographic tracing. *See* Pulse (*Sphygmograms*).

Postdiphtheritic. — Following diphtheria, especially as a sequel of the same, *e.g. postdiphtheritic paralysis. See* Diphtheria (*Complications*).

Postenteric.—Occurring after typhoid fever. *See* Typhoid Fever (*Sequelæ*).

Postepileptic.—Occurring after an attack of epilepsy. *See* Epilepsy (*Prognosis, Mental Degeneration*).

Postero-.—In compound words *postero-* means posterior to, and appears in such words as *postero-external, postero-lateral, postero-median, postero-temporal*, etc.

Postfebrile.—Following fever.

Postglenoid. — Situated behind the glenoid fossa of the temporal bone, *e.g.* the *postglenoid tubercle.*

Postgonorrhœal. — Occurring after gonorrhœa, *e.g.* postgonorrhœal arthritis, rheumatism, or synovitis. *See* Joints, Diseases of (*Gonorrhœal*); Rheumatism, Chronic (*Etiology*).

Posthemiplegic. — Following hemiplegia; postapoplectic. *See* Hemiplegia.

Posthitis.—Inflammation of the prepuce (Gr. *πόσθη*, membrum virile).

Postholith.—A preputial calculus.

Posthyoid.—Situated behind the hyoid bone.

Posthypnotic.—Following the hypnotic state, *e.g.* a *posthypnotic suggestion*).

Postmature. — Over-ripe, *e.g.* a *postmature fœtus* is one born after the full term of pregnancy.

Post-mortem Methods.

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This subject will be treated in two parts: the first describing the method of performing a "post-mortem" and of recognising the more important diseased conditions which may be met with; the second, describing methods of preserving tissues and of preparing them for histological or bacteriological examination. By the former one can hope to demonstrate only gross, macroscopic lesions, but even with this in view the examination must be carried out in a careful, systematic manner. In many cases a definite diagnosis can only be arrived at after a further histological or bacteriological investigation.

Part I

Method of performing a Post-mortem Examination

Rules of Procedure, and Precautions to be observed.—*Personal.*—Before commencing, examine the hands and wrists carefully for recent wounds.

Minute wounds which cannot otherwise be distinguished may be rendered manifest by the localised smarting produced by dipping the hands into weak solutions of ammonia or formaldehyde ("formalin").

If wounds exist, they must be sealed up with a solution of celloidin in equal parts of alcohol and ether. This is preferable to flexile collodion, as it adheres better to the skin. If this be not deemed sufficient protection, thin rubber gauntlets, which must be absolutely water-tight, may be worn.

Anoint the hands and wrists with antiseptic vaseline, and wipe the palms so that the knife will not slip.

If the hands be cut during an operation, the wound should be encouraged to bleed by squeezing it under a stream of *cold* water, or by suction, then washed with a strong antiseptic and sealed with celloidin. If, soon after, such a wound become hot and tender, indicating infection, it should be opened up, painted with

tinct. iodi. B.P., and a moist carbolic dressing applied. Immediate treatment often prevents grave consequences.

During the course of the operation keep the hands moist by an occasional dip into cold water, to which 1 per cent lysol, or 1·2000 corrosive sublimate may be added. Blood and discharges must never be allowed to dry on the skin.

On completion of the sectio, wash the hands thoroughly in *cold* water, preferably under a tap, using a nail-brush to remove all blood or discharges; secondly, in cold water with soap; then in hot water with soap. Finally, soak in 1·20 carbolic acid, 2 per cent lysol, or 1·1000 corrosive sublimate solutions, and wipe dry.

To deodorise the hands the best medium is a weak solution of "Sanitas" in water.

Operative and Technical.—In all post-mortem examinations system and cleanliness must be insisted on.

If possible, secure good light, preferably daylight.

In making incisions, always cut *away* from, never towards, the other hand, or the hands of an assistant, and in laying down a knife place it at some distance.

As a rule make long sweeping cuts with the belly of the knife, drawing it through, rather than pressing it into the tissues.

Use sharp knives. Blunt knives are dangerous, as more force is required, and this is apt to make the knife slip, with the attendant dangers.

If an organ is worth preserving make as few incisions into it as possible, consonant with the attainment of the desired information.

If a bacteriological examination be necessary, inoculations should be made at once into suitable media, or the organ conveyed whole to the laboratory, so that its investigation may begin as soon as possible.

Instruments and Accessories required

Knives:—

One strong "cartilage" knife, for making the skin incision, and cutting through the costal cartilages.
One or more strong scalpels.
One curved, prob-pointed bistoury.
One long amputation or "brain" knife for making sections of organs.
Bowel scissors.
Sharp pointed scissors.

In place of the two last, one pair of scissors may be used, with blades two to three inches long, one probe pointed, the other sharp.

A strong saw; cutting bone-forceps; strong dissecting forceps; chisel and mallet.

Two strong needles curved in half their length.

Strong thin cord or "Dutch" twine.

The above are essentials, and they or their equivalents are to be found in the armamentarium of every practitioner.

The following may be added:—

Lion forceps; syringe with fine nozzle; measure glass; cones for measuring the diameters of orifices; measuring tape or foot-rule; balance and set of weights.

Method of performing a Post-mortem Examination.—The following description applies to cases in which the whole body is to be examined. Frequently the examination is only partial, as necessitated by the requirements of diagnosis or the wishes of the relatives.

After laying out the instruments likely to be required, and, if in private, disposing sheets or newspapers so as to catch any blood or discharges, the first thing to be done, after noting the name, age, date of death and post-mortem, is to make an external examination.

External Examination

In special cases measure the length of the body and its girth at the shoulders. It is seldom necessary to weigh the body.

Note muscular development and state of nutrition.

Rigor mortis.—It is well at this stage to ascertain the presence and extent of rigor mortis, as subsequent movement of the body may abolish it in the neck and limbs.

Rigor mortis is a certain sign of death, and is caused by the coagulation of the myosin of the muscle fibres. Under ordinary circumstances it appears about five or six hours after death in the muscles of the head and neck, extending later to the trunk, then to the upper and lower extremities. When decomposition commences it passes off in the same order, from above downwards. Its onset and duration depend on several factors, such as temperature of the air, cause of death, and age of the individual.

In dry cold weather its duration is greater, the decomposition which abolishes it being delayed.

After exhausting illnesses, and those in which numerous convulsive muscular contractions have occurred, its onset is earlier than usual, and its duration short.

It begins earlier and passes off sooner in the young than in the well-developed adult.

It is often slight after death from sepsis, and passes off early.

It is practically never absent, though it may be almost imperceptible, for example, in puny, emaciated infants.

Examine the *surface* of the body from the head downwards to the feet.

Examine the *scalp* for cuts, bruises, and prominences, such as wens or tumour-growth. Note the condition of the *eyes* as to prominence; subconjunctival hæmorrhage, pointing to fracture of the bones forming the orbital cavity;

inequality of the pupils.—These are usually of medium size, and equal.—Note the presence of eruptions on the face and around the mouth.

Note escape of blood or discharges from the nose, mouth, or ears. Bleeding from the ears is usually present in fracture of the base of the skull. If the blood come from the nose or mouth it may signify rupture of an aneurysm into the air passages or œsophagus; hæmorrhage into the stomach; hæmorrhage from the mucous membrane of the nose, nasopharynx, or mouth; or, fracture of the base of the skull.

Discharge of pus from the ear would draw attention to the condition of the middle ear and mastoid cells, and suggest the possibility of secondary intra-cranial suppuration.

Passing from the head to the *trunk* and *extremities*, one has to note the presence of wounds and scars, with their probable date, œdema of the surface, congenital marks and malformations, discolorations, disfigurations from accident or otherwise.

Discolorations may be of the following varieties: tattoo marks; pigmentation in connection with chronic ulcers, or the result of the occupation of the deceased; bruises and ecchymoses; hypostatic lividity; and discolorations due to decomposition. The last three are of importance.

Ante-mortem bruising is distinguished by a permanent discoloration which cannot be removed by pressure. If the part be incised the blood is seen to have escaped from the vessels, and to lie in the surrounding tissues.

Post-mortem lividity is a reddish or purplish discoloration occurring usually in dependent parts of the body, for example in the skin of the back when the body is lying with the face upwards. Over bony points the skin is compressed and lividity is absent, and it can be removed from other parts by pressure. It is caused by the blood-serum containing dissolved blood-pigment soaking out from the vessels into the surrounding tissues. As a general rule lividity is greatest in cases where the blood remains fluid, for example, in pronounced septic cases, or in cases of asphyxia due to accident or resultant on other diseases. In septicæmia and other morbid conditions in which there has been breaking down of the blood corpuscles setting free the pigment, the lividity may be general, and the superficial veins are distinctly mapped out, forming a close purplish network.

In ordinary cases, after twenty-four to thirty-six hours, signs of *decomposition* appear over the abdomen as a greenish discoloration of the skin. This is due to the action of sulphuretted hydrogen and ammonium sulphide upon iron compounds, black sulphides being produced in the subcutaneous tissues. This discoloration appears earlier over the site of suppurations in the tissues or cavities of the body.

The surface of the abdomen in women who have borne children, or in great distension from other causes, and that of the thighs and mammæ in fat women, may show numerous small silvery scars — "striæ gravidarum" or "lineæ albicantes"—due to stretching of the skin.

Enlarged glands in the neck, axilla, or groin may be felt readily through the skin.

In *rickets* in children the "rickety rosary," formed by thickening at the costo-chondral articulations, is readily made out, and at the same time the ends of the long bones are enlarged from the excessive proliferation of the cartilage cells at the epiphyses.

Examination of the Body Cavities

The Thorax and Abdomen are usually examined before the Head. To open them a median incision is made through the skin and superficial structures from the cricoid cartilage to the symphysis pubis, passing to one side of the umbilicus. The knife must be held with a full grip—never as one holds a pen—and the arm kept rigid, the movement being at the shoulder rather than at the wrist or elbow, and the cut being made with the belly of the knife.

After making this preliminary incision dissect down carefully in the epigastrium until on cutting through the peritoneum the liver comes into view; then, inserting two fingers of the left hand downwards into the abdomen along the line of the skin incision, draw the abdominal wall upwards and cut downwards between the fingers, opening up the cavity completely. If more room be required below, cut across the insertions of the recti abdominales from the peritoneal aspect.

Take hold in turn of the edges of this incision, and, pulling the flaps outwards, with bold sweeps of the knife reflect the whole of the superficial soft parts from the thoracic walls and costal margins as far as the nipple lines.

On first opening the peritoneum gas may escape from the cavity, indicating perforation of the intestine; escape of large quantities of fluid points to ascites.

Note *œdema* of the subcutaneous tissue, and any morbid change in the muscles—pallor, fatty or hyaline degeneration.

Note the position and relationships of the abdominal contents; the height of the *diaphragm* —normally on a level with the fourth right and the fifth left ribs; the relationship of the edge of the *liver* to the costal margin, crossing it normally in the nipple line at the level of the ninth right costal cartilage. This relationship may be altered by change in the size of the liver, or by alteration in the height of the diaphragm caused by increase or decrease in the volume of the thoracic or abdominal contents. Note any irregularity of the surface of the liver caused by cirrhosis, syphilitic cicatrices, or tumour-growth.

Passing downwards, note the extent of *stomach* wall visible, the amount normally visible not

exceeding six to nine square inches. Note the position and appearance of the *colon* and *great omentum*, the latter being greatly thickened and shortened in certain cases of tubercular peritonitis, and showing a varying amount of fat even in health. Then, raising the omentum, along with the transverse colon, over the costal margins, look for general or partial distension or contraction of the *intestines*, and if present find out whether the conditions have relation to a stricture or other obstruction—tumour or hernia—or be simply due to irregular peristalsis. Lymph, blood, pus, or fluid fæces may be present between the coils of intestine, the last indicating perforation of its walls. The coils of intestine near the gall-bladder usually show post-mortem staining with bile.

Diverticula may be found in connection with the small intestine, for example *Meckel's*, connected to the ileum eighteen inches to three feet above the ileo-cæcal valve. *False diverticula* are simply the exaggerated pouchings of the large intestine met with in cases of fæcal loading.

Peritonitis.—The only indications of peritonitis may be general congestion and dulling of the surface.—In *hypostasis* the posterior coils are congested, and the surface bright.—In peritonitis there is usually, however, a variable amount of lymph lying on the surface, or forming prismoidal collections between adjacent loops of intestine. The lymph may be simple, puriform, or may have organised, producing recent or old, general or localised, adhesions between coils of intestine, or between visceral and parietal peritoneum.

The older these adhesions are the less easily are they broken down. In general tubercular peritonitis they contain small or large caseous nodules.

Localised peritonitis may occur on the surfaces of the liver or spleen, round an appendicitis, or in the pelvis in the female.

As a result of old peritonitis bands of fibrous tissue may pass from one part of the peritoneal surface to another, forming loops in which internal herniæ may form.

Intussusception is a condition in which a portion of intestine is drawn downwards into another part, forming a firm, sausage-shaped mass which, if unrelieved, becomes gangrenous. The entering bowel becomes adherent by peritonitis to the orifice of the ensheathing bowel.

"*Agonal*" *intussusceptions* are occasionally found in children. They are usually multiple, only occur in the small intestines, and are distinguished by the absence of inflammation, and by the fact that the entering bowel can be readily disengaged.

Volvulus is a condition in which a sigmoid flexure with long meso-colon becomes twisted on itself, causing obstruction.

In *gangrene* of the intestine—for example, in strangulated hernia—the bowel becomes dark, livid, or greenish brown, and dull on the surface.

Note thickening, localised or diffuse, in the mesentery—the former due to enlarged glands, the latter to tuberculosis, sarcoma of glands, or mesenteric abscess.

Before removing the abdominal organs it is well at this stage to open the thorax in order to examine the serous cavities, and to make a general examination of the lungs and heart, particularly as to volume. If this be omitted the blood will drain away when the abdominal vessels are cut across, and the relationship of the thoracic organs will be disturbed.

For directions refer to the method of opening the thorax described later (*see* p. 204).

This being done, remove the abdominal viscera in the following order, which experience has shown to give the most satisfaction.

Removal of Viscera.—In the first place remove the *small intestine*. Taking up a coil, separate, with a sharp knife, the mesentery at its attachment to the bowel, and continue this until the whole small intestine is free. Then cut it across between double ligatures placed at the upper end of the jejunum and three inches above the ileo-cæcal valve.

It may be examined at once.

In miliary tuberculosis the serous surface presents numerous grey pin-head tubercles. These may be "general" throughout the peritoneum, or localised to the base of ulcers of the Peyer's patches, which shine through the wall as dark purplish areas. They must not be confounded with miliary cancer nodules, which are usually larger, often bile-stained, and are usually surrounded by a narrow zone of congestion.

Next, placing the intestine in a sink or large basin, remove the ligatures and flush it with a stream of cold water, and with the scissors, the blunt blade being inserted into the bowel, cut it open along the line of attachment of the mesentery. The Peyer's patches are thus uninjured, being placed opposite to that line.

Examine the mucous membrane for congestions and hæmorrhages, catarrhal enteritis, ulceration—typhoid, tubercular, or follicular—perforations, or for waxy degeneration in cases of chronic suppuration and phthisis pulmonalis.

In *catarrhal enteritis* the mucous membrane is congested, especially over the valvulæ conniventes, thickened and covered with tenacious mucus.

In *typhoid ulceration* the whole of the Peyer's patch is affected, hence the ulcer is usually longitudinal, with sharply cut, thin edges. The solitary glands are also affected. The floor of the ulcer is formed of the inner circular muscular coat.

In *tuberculosis* the ulcer in the Peyer's patch

is irregular, with sinuous, worm-eaten outline and thickened edges. It tends to spread round the bowel like all other chronic ulcers. The floor is irregular and caseating; the mesentery is usually thickened and shortened. Miliary tubercles can always be made out under the serous surface.

In health little if any decomposition occurs in the small intestine, its functions being mainly digestive and absorbent. The fæces are fluid or semi-fluid.

The LARGE INTESTINE may now be removed. Placing the great omentum on the stretch detach it from the transverse colon. Then, commencing at the caput cæcum detach the colon and upper part of the rectum by cutting and tearing through their loose attachments. In removing the ascending part, be careful not to injure the kidney, or open into the duodenum to which it is closely applied.

Before dividing the rectum press its contents upwards, pass a ligature round its lower part and cut below the ligature. This procedure prevents soiling of the pelvis with fæces.

Wash out the large intestine, and cut it open along one of the longitudinal muscular bands. Open up the caput cæcum and slit up the *appendix vermiformis*.

The orifice of the appendix normally admits a probe; it may be constricted or obliterated. The cavity may be distended with mucus or contain foreign bodies or concretions resembling foreign bodies. Its walls may be ulcerated, gangrenous, or perforated. Its relations vary greatly under normal conditions.

Catarrh, with congestion of the ridges of the mucous membrane, is commonly seen in the large intestine. In infantile diarrhœa the intestine may present no morbid appearance. *Dysentery* is characterised by fibrinous deposit upon or in the mucous membrane, and sloughs may form and separate. *Tubercular ulceration* may occur somewhat similar to, but more extensive than in the small intestine.

Syphilitic ulcers are characterised by their serpiginous outline, undermined edges, and tendency to cicatrize. In *fæcal* accumulation—for example, behind a stricture—ulcers may be found in the colon.

Tumours of the large intestine may be non-malignant; for example, fibrous or myxomatous polypi and adenomata. They are more often malignant, tubular cancer being the most common, and occurring usually in the cæcum and at the flexures. The cancers proper are occasionally met with in the rectum, rarely elsewhere. Squamous epithelioma may occur at the anus.

Mesentery.—Examine for enlarged or tubercular glands. Apart from secondary metastases the only tumour to be looked for here is lympho-sarcoma, commencing in the retro-peritoneal glands. Other tumours are rare.

The *Spleen* may now be removed. Its normal average weight in the adult is 6 oz. Its average length is from 5 to 6 inches; its breadth from 3 to 4 inches, and its thickness a little over one inch.

Its capsule may show adhesions or fibrous thickening.

In atrophy of the spleen the capsule becomes wrinkled and loose.

In chronic venous hyperæmia it is enlarged in all directions, firm and cuts crisply; its cut surface is flat, of a dark plum-colour, and shows numerous fibrous points and bands.

In acute hyperæmia the organ is usually enlarged, soft, and on section the pulp bulges, is soft and readily scraped away or washed away with a gentle stream of water. This is the "diffluent" spleen. It indicates pronounced septic infection.

In waxy change the Malpighian bodies may be specially affected—"sago" waxy—or the change may be "diffuse" throughout the pulp. These two varieties are usually combined to a greater or less extent. Application of a weak solution of iodine colours the waxy parts a deep mahogany colour.

℞	Iodine . . .	1 part.
	Potass. iodide . .	2 parts.
	Water . . .	100 parts.

In general tuberculosis, miliary or caseous tubercles may be met with in the spleen.

The arteries of the spleen being of the nature of "end-arteries," blockage of them, for example by an embolus, results in the production of a "hæmorrhagic infarct," which, if the patient survive, becomes decolorised. This is usually wedge-shaped with its base to the surface, and projecting slightly upon it. Still later, the infarct becomes surrounded by contracting scar tissue, forming a groove on the surface.

Tumours are rarely met with in the spleen.

Gummata—tough necrotic areas of the consistence and colour of wet wash-leather are occasionally met with. The spleen may be greatly enlarged in leucocythæmia, ague, and occasionally in rickets.

Small accessory spleens or *spleniculi* are often present near the spleen, resemble it in structure, and share its morbid changes.

Remove the *liver*, *stomach*, *duodenum*, and *pancreas* in one mass. By so doing the continuity of the bile passages is conserved.

Cut through the suspensory ligament of the liver, freeing the organ from the diaphragm, then drawing it downwards, isolate and ligature the œsophagus as far up as possible. It is then easy to separate the organs above mentioned from the posterior abdominal wall, care being taken not to injure the suprarenal capsules, particularly the right one, which lies close to the lower surface of the liver.

Placing the organs in their natural position,

examine, firstly, the Stomach and Duodenum. Note the size of the stomach and presence of thickenings in its walls. It is dilated in stricture of the pylorus, small in stricture of the cardiac orifice or in general scirrhus of its walls. Remove the ligature and allow the contents to flow out into some receptacle.

These may be food, mucus, bile-stained material, clotted blood, or "coffee-grounds."

For investigation of poisons, etc., refer to article on "Toxicology."

Wash out the STOMACH AND DUODENUM, and slit them open, the former along its greater curvature, the latter along its convexity. If a stricture be present, preserve it intact.

Examine the *mucous membrane* of the stomach. Distinguish *post-mortem digestion* from *gastric ulceration.* The former occurs at the dependent part of the wall, hence usually at the cardiac end posteriorly. The walls become gradually thinned from digestion of the coats from within outwards, and if an opening exist in the centre of the thinned area, it has thin, frayed-out edges.

Gastric ulcers are rounded, "punched out"; if chronic they possess thickened edges, and are most commonly met with near the lesser curvature on the posterior wall towards the pylorus. Their base is formed by submucous or muscular coat, or by some subjacent organ, such as pancreas, which the ulceration has exposed.

Hæmorrhage may come from a gastric ulcer: from ulceration over varicose veins at the lower end of œsophagus—for example, in cirrhosis of the liver: from rupture of an aneurysm: from ulceration of a cancer: or the blood may have been swallowed.

In *acute gastric catarrh*, the mucous membrane is congested, swollen, œdematous, and covered with tenacious mucus. In *chronic gastric catarrh* the walls are thickened, the rugæ are prominent, and numerous rounded, mammillated prominences may be present.

Tumours.—Papilloma at the cardiac orifice; tubular, hard and soft cancers at the orifices or in the body of the organ are met with. Tubular cancers are usually flat, the edges raised and slightly overhanging, their centres ulcerated. Hard cancers show great and firm thickening of the mucous and submucous coats, and hypertrophy of the muscular coats; they tend to contract. Soft cancers are characterised by their abrupt, thick, rampart-like edges, their fungation, soft consistence, and great tendency to ulcerate in the centre. Carcinoma of the stomach may undergo colloid degeneration. Squamous epithelioma may spread from the œsophagus to the cardiac end of the stomach.

Duodenum.—Ulcers similar to those in the stomach are not unfrequently met with in the first part.

Catarrhal inflammation shows characters similar to catarrh of the rest of the alimentary tract.

Stricture may occur at the entrance of the biliary and pancreatic ducts; and tumour growth, non-malignant or malignant, may also occur there, the latter often as an extension from the head of the pancreas.

Lay the liver on its upper surface and arrange the duodenum so that no kinking of the common bile-duct can take place, and by pressure on the gall-bladder endeavour to force bile downwards into the duodenum. If it do not pass, lay open the bile-ducts carefully throughout their whole length, and examine for stricture, compression by scar tissue or tumours, or for the presence of calculi blocking them.

Next lay bare the *pancreas* from the front; note its consistence, and any pronounced enlargement, then slice it longitudinally to determine whether any tumour-growth be present, and transversely to determine the amount of fibrous tissue present. Note whether it be softened and hæmorrhagic (hæmorrhagic pancreatitis) or show suppuration or gangrene. It is not necessary in ordinary cases to expose its duct. Occasionally the pancreas is found ruptured in cases of abdominal injuries. The pancreas weighs from 3 to 4 ounces in the adult.

The gastro-hepatic omentum may now be divided and the liver separated and examined.

THE LIVER.—The normal average weight of the liver is 3 lb. 5 oz. in the male, and 2 lb. 12 oz. in the female. It measures transversely from 10 to 12 inches, antero-posteriorly from 6 to 8 inches, and its greatest thickness is from 3 to 5 inches. Relatively it is larger in the child than in the adult.

It is of a chocolate colour, and its substance can be distinguished through the capsule, which is thin, smooth, and glistening.

The lobules, which vary in size from $\frac{1}{16}$ to $\frac{1}{20}$ of an inch, can be faintly distinguished. On the cut surface the openings of numerous large veins are seen. When laid on a plane surface the organ flattens out.

Morbid Conditions.—The capsule may show localised or general fibrous thickening. The surface may show transverse or antero-posterior grooves, the result of compression, for example by corsets. The organ may be displaced from the same cause. It may be ruptured from injury.

Degenerations and Infiltrations of the liver.

Cloudy Swelling.—This usually occurs in death with high temperature. The organ is paler than normal, the capsule tense, the cut surface smooth and glistening, with the individual lobules somewhat enlarged and outlined by a rather indefinite pale zone.

Fatty Infiltration.—The organ is enlarged, brown or orange in colour, the consistence lessened, the individual lobule much increased

in size, and outlined by a distinct pale or yellow zone. In fatty hypertrophic cirrhosis the organ is greatly enlarged, and may be of a canary yellow colour throughout.

Waxy or Amyloid Disease. — The organ is greatly enlarged, its consistence is firm, and it preserves its shape when removed from the body. It is pale, translucent, and of a pinkish grey colour. The lobules may be outlined by a yellow zone of fatty infiltration. The edge of a section remains sharp. The lobule is greatly increased in size, measuring $\frac{1}{10}$ to $\frac{1}{6}$ of an inch, and the intermediate zone is marked out as a distinct, greyish, translucent ring. Application of the iodine test (*vid.* spleen) renders the waxy change very evident.

Fatty Degeneration. — The organ is smaller than normal, the capsule apparently thickened, and is wrinkled. The organ is soft, very friable, of a dull orange colour; the lobule may show a ring or spot of a yellower colour, according as the central or peripheral zone is affected. Either zone may show the change, the former quite as often as the latter.

In *chronic venous hyperæmia* the central zone of the lobule is marked out as a dark brown spot. This gives a mottled appearance, "nutmeg liver," to the organ. It is much enlarged and may be firmer than normal. The condition results from chronic valvular disease of the heart.

Multilobular Cirrhosis.—In this condition the surface of the liver is covered with rounded projections, varying in diameter about from one-twentieth of an inch to about one-fifth of an inch. Separating them is a network of shallow grooves, caused by indrawing of the capsule by scar tissue formed along the portal vessels. On section a similar network is seen of pinkish grey colour separating opaque orange coloured islets of liver tissue. The organ is usually diminished in size, but may not weigh less than normal. The condition gives rise to ascites and chronic venous hyperæmia in the portal venous system.

Monolobular Cirrhosis.—The organ is usually increased in size, the capsule smooth or only slightly wrinkled, and the newly formed fibrous tissue is made out with difficulty, as the network it forms is finer, enveloping individual lobules.

In *acquired syphilis* gummata may form in the liver. *Congenital syphilis* is associated with a diffuse fibrosis of the liver.

In *acute yellow atrophy* the organ is much lessened in size and flabby, the capsule being wrinkled. On section it shows alternating yellow and rusty red patches.

Suppuration.—Tropical abscesses are usually single and of large size, and the liver substance shows degenerative changes.

Abscesses from other causes are usually small and multiple; they may be part of a general *pyæmia*, of a pyæmia confined to the portal system, or may result from a suppurative inflammation of the bile-ducts (*cholangitis*).

Tuberculosis. — *Miliary* tubercles occur as minute grey points best seen under the capsule. *Caseous* tubercle occurs as small yellow points or larger bile-stained softening areas.

Parasites.—The most common parasite is the cystic stage of the Tænia echinococcus—the "hydatid cyst."

The cyst wall is formed of thin laminæ, of a material resembling the boiled white of a duck's egg. It is smooth and glistening on its outer surface, but granular on its inner surface. It usually contains secondary cysts. In one variety the secondary cysts form in the tissue round the primary cyst, producing the "multilocular" hydatid.

The important *new-growths* are malignant, and may be primary or secondary. The former may be single, the latter are practically always multiple. They may be of the nature of cancers —hard, soft, or tubular, non-pigmented or pigmented, or they may be sarcomata.

In *hard* (scirrhus) cancer the nodules are usually of firm consistence, and have a sharp outline. They tend to retract rather than degenerate towards their centre, so that an appearance of hollowing or "umbilication" may be produced on the surface. This is, however, by no means confined to the hard cancers.

In the *soft* cancers (medullary or encephaloid) the tumour is usually rounded, soft, and spongy. It tends to degenerate early in its centre.

In *tubular* cancer (malignant adenoma) the nodules are usually numerous, rather irregular in outline, on the surface showing more variegation of colour than the others, for example, pink, yellow, and creamy areas. In this variety, especially, the liver is greatly increased in size.

The *sarcomata* are usually secondary, may be cerebriform in appearance and consistence — round-celled sarcoma—or firmer and fasciculated —spindle-celled sarcoma.

The *pigmented* (melanotic) tumours are necessarily secondary, and are readily distinguished by their sepia coloration. If the primary tumour be discernible, this will settle their epithelial or connective tissue origin. Usually, however, microscopic examination is necessary.

Resumé

The liver is *diminished* in size in—

Fatty degeneration, simple atrophy, acute yellow atrophy, late multilobular cirrhosis, and often in cancer of the pyloric end of the stomach.

It is *increased* in size in—

Cloudy swelling (slightly); fatty infiltration, unilobular cirrhosis especially with fatty change; waxy disease; chronic venous congestion; cancer; occasionally in early multilobular cirrhosis and in obstruction of common bile-duct. It is also enlarged, but irregularly, in abscess formation, and in presence of hydatid cysts.

The substance is *paler* than normal in—
Cloudy swelling; fatty changes; waxy disease; cirrhosis.
It is *darker* than normal in—
Chronic venous congestion; in pigmentary conditions and tumours.
Its consistence is *softer* in—
Fatty changes; acute yellow atrophy.
Its consistence is *firmer* in—
Cirrhosis; waxy disease; late stages of chronic venous congestion; fibrosis from syphilis or other cause.

The only organs now left in the abdomen are the *kidneys* and *suprarenal capsules.*

The latter are not usually examined closely, unless for some reason or other attention is specially directed to them.

They may show *waxy* degeneration along with other organs, where chronic suppurations or tertiary syphilitic conditions exist. They are the seat of tubercular *caseation* in Addison's disease, and occasionally show tumour-growth, usually secondary. In cases of toxæmia the medulla may become so soft that, in removal from the body, it breaks down, and the organ comes to resemble a cyst bounded by the more resistant cortex.

The *Kidneys* must always be carefully examined.

To remove a kidney, cut through the peritoneum at its outer margin and peel it off the surface, then raise the organ out of its bed, separating the structures surrounding it until it is attached only by its vessels and ureter. Cut these across at some distance from the hilum, noting whether any fluid escapes from the ureter, and if so, its character, whether clear or turbid. If the ureter or ureters be dilated, it is advisable to remove the whole urinary tract, including part, at any rate, of the corpus spongiosum urethræ, particularly if stricture of the urethra be suspected.

After examining the surface and noting abnormal firmness of perinephric fat, presence of perinephric abscess, or congestion of the surface, cut it open from the outer margin inwards towards the hilum, being careful not to injure the hand holding it. Then examine in order—

Capsule.
Cortex.
Medulla.
Renal pelvis and ureter.

In normal conditions in the male the left kidney weighs, on an average, 5½ ounces, the right kidney 5¼ ounces, in the female being about half an ounce lighter. The left is usually longer and narrower than the right, but this distinction does not always hold, and one has to determine the side of the excised kidney by the position of the structures entering and leaving its hilum; the vein is in front, the artery in the middle, and the ureter behind and at its lower part.

If fat surround it, it should separate easily. The capsule is thin, translucent, and when a section of the organ is made should strip readily. On the cut surface the cortex should bear to the medulla a relationship of about one to three, and should be of a dull, brownish colour, but paler than the medulla.

In children a lobated arrangement is apparent up to about the third year, and on section the interpyramidal cortex may also be separated into two, each part in connection with its own pyramid. This condition may persist—"fœtal lobulation."

The two kidneys may be connected at their lower part by kidney substance or by a fibrous band. This is the "horse-shoe kidney." The ureters always pass in front of the connecting bridge. The malformation does not interfere with function.

If one kidney becomes functionless the other undergoes compensatory hypertrophy.

A uniform cystic transformation exists in the congenital cystic kidney.

If a sharply circumscribed area of an otherwise functionating kidney be cystic, it usually indicates an adenomatous new-growth.

If scattered cysts exist in a firm, functionating kidney, they are retention cysts, due to disease (chronic interstitial inflammation) of the organ, or to other causes obstructing the tubules.

General Characters of Disease in the Kidneys.—A soft, flabby kidney indicates parenchymatous change.

A kidney of firm consistence indicates an interstitial change.

A *very* easily stripped capsule indicates a parenchymatous change.

A thickened and adherent capsule indicates interstitial change of some standing. In removing it, portions of kidney substance adhere to it.

A thickened subcapsular cortex usually indicates parenchymatous change, particularly if pale, or patchy, or yellow.

A thickened inter-pyramidal cortex is usually present in interstitial inflammation.

An atrophied cortex, associated with adherent capsule, indicates chronic interstitial inflammation.

Marked increase of fat round the pelvis indicates atrophic changes in the kidney such as occur in chronic interstitial nephritis, or arterio-sclerotic atrophy.

Yellow spots in the kidney substance, surrounded by a zone of congestion, are suppurative in origin.

Grey or yellow spots, without a zone of congestion, are tuberculous in origin.

Distinct dilatation of the pelvis of the kidney —*hydronephrosis*—occurs only from obstruction below in the urinary passages. Unilateral hydronephrosis results from obstruction or stricture of the ureter corresponding. Bilat-

eral hydronephrosis occurs most commonly in stricture of the urethra or phymosis.

Hydronephrosis is to be distinguished from *scrofulous kidney*. In this large areas of the kidney have caseated and broken down into the pelvis, so the remains of the kidney bound an irregular cavity with diverticula, as in hydronephrosis. Here, however, the cavities are rough-walled, lined with caseous material, whereas in hydronephrosis the walls are smooth, lined with mucous membrane. The two conditions may be conjoined.

Waxy or Amyloid Disease.—Here the history of the case helps greatly to a diagnosis.

The waxy change occurs along with other parenchymatous or interstitial changes in old chronic suppurations and excavations of pulmonary tuberculosis, and in tertiary syphilis. The organ is enlarged, sometimes very greatly, as when the condition accompanies parenchymatous inflammation. It is soft, pale, of a light, pinkish grey colour, with streaks and patches of yellow in the cortex. The tips of the pyramids are pale, the boundary layer still remaining the darkest part of the organ. In the cortex the Malpighian bodies stand out distinctly as translucent grey points, and the iodine solution displays well the waxy glomeruli and straight vessels.

When there is accompanying or pre-existent interstitial change the organ is slightly enlarged, firmer than normal, its general appearance on section resembles that of the other variety, but the patchy pallor exists throughout the organ, the boundary layer being slightly, if at all, redder than the rest of the surface.

Infarcts of Kidney.—These are localised areas of necrosis, simple or suppurative, caused by embolic blocking of an interlobular artery. The area corresponds to the portion supplied by the artery. They are pyramidal in shape, the base towards the capsule, and slightly raised above its general surface. They are pale and surrounded by a hæmorrhagic zone. Organised infarcts are seen as scars, causing a localised adhesion and indrawing of the capsule.

Calculi, irregular and brownish, may occur in the pelvis of the kidney or in the ureter at its lower end, where it enters the bladder. This is the narrowest part of the tube.

With the indications above mentioned the more common morbid conditions of the kidney and ureter can be, at all events approximately, diagnosed with the unaided eye.

Abdominal Pelvis.—In peritonitis lymph or pus may be more evident in the pelvis than elsewhere in the abdomen, having gravitated there.

Male Pelvis.—Examine the pouch of Douglas for effusions, pus, or fluid fæces which have escaped from a perforation of the bowel.

Bladder. — Note the state of the bladder, distended or empty. Try to express its contents through the urethra, to show whether this be patent or strictured. The bladder may be catheterised or opened *in situ*, and the condition of the urine and inner surface of the bladder ascertained. If the urine be decomposing there is probably cystitis.

Cystitis is indicated by congestion of the surface and rugæ, which may be covered with tenacious mucus.

Examine the walls for hypertrophy. In this condition the muscular rugæ are very prominent. Do not confound this with the apparently thickened and rugose walls of the contracted bladder.

Remove the bladder, separating the urethra from its connections to the arch of the pubis, and cutting it across at the posterior part of the bulbous portion. Slit up the bladder, prostate, and urethra along the anterior surface. Examine the prostate for hypertrophy, tumour-growth, suppuration, tuberculosis, or calculi.

In cases which demand their examination expose the *vesiculæ seminales* on the outer posterior wall, and make sections. They present a honeycombed appearance, and contain normally a clear, viscid, slightly yellow fluid. They may be thickened and full of pus, or may show tuberculous change.

The *rectum* is usually removed along with the bladder, and should be slit up along its posterior wall. It may show congestion and catarrh, or tumour growth — polypi (adenomatous), the cancers proper, or squamous epithelioma. Occasionally non-malignant stricture is met with, for example, after syphilitic ulceration (in women).

Remember that in the rectum normally there are two projecting semi-lunar folds of mucous membrane, which must not be mistaken for new formations.

Female Pelvis.—Note adhesions from old peritonitis. These are often seen crossing the pouch of Douglas.

In all cases in women in which malignant tumours occur higher up in the abdomen, particularly in the liver, look for possible primary growth in the uterus or ovaries.

The body of the uterus in the adult projects about 3 inches behind and $2\frac{1}{2}$ inches in front upwards into the pelvis, the peritoneum thus covering it completely on its posterior surface, and in great part on its anterior surface. It is about 2 inches broad at its fundus and about an inch thick. The cervix uteri is not visible from the pelvis.

The uterus may be enlarged and project into the abdomen in pregnancy and in internal tumour-growth. Generally speaking, these conditions may be distinguished by the position of the Fallopian tubes, which leave the uterus high up in the former case, and low down in the latter case. Fibroids of the uterus usually

cause irregular enlargement of the body of the uterus, and if sub-serous, may form large projections from its surface.

In advanced malignant new-growths originating in the cervix, the lower part of the uterus may be destroyed, leaving an irregular cavity bridged across transversely by the remains of the fundus and the Fallopian tubes attached to it.

The *Fallopian tubes* pass outwards from the cornua of the uterus along the upper borders of the broad ligament. They vary in length, and their outer extremity, which is fimbriated, turns more or less backwards. The outer end may be occluded, leading to dilatation of the tube with watery, purulent, or hæmorrhagic fluid. Enlargement of the tube may be due to salpingitis, tubal gestation, or tumour-growth. The internal lining membrane is normally thrown into numerous longitudinal folds, which are most evident towards the outer extremity. A small pear-shaped, thin-walled cyst, the hydatid of Morgagni, is frequently seen attached to the fimbriated end, and is not abnormal.

The *ovaries* are attached by a fold of peritoneum to the posterior surface of the broad ligament. They are usually about the size of an almond in its shell. In old age they become hard, atrophied, and scarred on the surface.

In *peritonitis* they may be covered with lymph, which may be purulent. If inflamed, they are enlarged, soft, and congested, and may contain abscesses.

New-growths may be solid—cancer, sarcoma, fibro-myoma—or cystic.

The cystic growths are important. They may be of great size and weight, projecting far up into the abdomen.

They may be of the *compound cystic* variety, the cysts containing a glairy, viscid fluid, or the cysts may present papillomatous ingrowths, or similar projections on their outer surfaces. In these two varieties nothing apparently is left of the ovary. The Fallopian tube may not be greatly altered.

Large *single cysts*, over which the Fallopian tube is stretched, develop from the *parovarium*—a fœtal structure lying between the ovary and Fallopian tube. In this case the flattened ovary can usually be made out on the lower wall of the cyst.

Dermoid cysts, full of fatty material, and containing hair, teeth, and other representatives of organs, or parts of organs, may develop in the ovary. They are usually smaller than the cysts already described.

Graafian Follicle.—A Graafian follicle, after rupture in menstruation, appears as a brownish rounded cyst not larger than a pea, the colour being due to hæmorrhage into the cyst. Later the colour changes to yellow—corpus luteum—from organisation of the clot and fatty degeneration of the epithelium. Later still the fibrous tissue formed contracts, and a scar results. The corpus luteum of pregnancy is larger, and may remain of some size until the end of pregnancy, whereas that of menstruation remains for a few months only. Small cysts of the ovary may result from simple "dropsy" or distension of a Graafian follicle.

To examine the *Uterus* further, it is advisable to remove it from the body along with a portion of the vagina. The method of procedure is practically the same as followed in removal of the male pelvic organs, cutting through the vagina low down, on the same level as the rectum and urethra.

Open up and examine the rectum as in the male.

If normal, dissect it away from the vagina. Then, if the bladder be still attached, slit up the *vagina* in the middle line posteriorly.

Note presence of lacerations or ulcerations of its surface. If discharges be present note whether they come from vagina or os uteri. Note amount of projection of the *cervix uteri*, which is proportionately longer than normal in "conical cervix" and in infants.

External Os.—The nulliparous os is round and smooth; the parous os is transverse and fissured. The mucous membrane surrounding may be red and rough—"erosion"—or protrudes as a soft red mass, which may be mistaken for malignant disease. Occlusion of the os is rare.

Extensive destruction of the cervix occurs usually from malignant disease. *Cancer* may be squamous-celled, starting from the vaginal portion of the cervix, or tubular, starting from the mucous membrane lining the cervix, or from the glands opening on its surface. The surface of the tumour is usually very irregular, ulcerated, or warty.

Non-malignant *polypi* of the cervix project through it; they are glandular and small in size. *Fibroids* occur rarely in the cervix, though pedunculated uterine fibroids may project through it.

Open up the cervix and uterus in the middle line, and extend the incision into the Fallopian tubes.

Note the consistence and thickness of the uterine walls.

The normal cervix is fusiform in shape and rugose on its inner surface. Distinction of cervix from uterus is lost in cases of recent delivery. It may be elongated or hypertrophied.

The body of the uterus may show rounded, encapsuled *fibro-myomata*—firm bodies, on section fasciculated and glistening. They may reach a great size, and project internally or externally. They may become pedunculated. The *uterine polypus* is usually a pedunculated submucous fibroid.

Malignant disease of the uterus usually

spreads from the cervix, though sarcoma and tubular cancer may commence at the fundus.

In *puerperal fever* the inner wall is shreddy and covered with foul discharge. Ulcers with sloughy base may be present, or abscesses may be present in the uterine wall. The ovarian veins may contain purulent thrombi.

The remaining structures on the posterior abdominal wall must now be examined. The *aorta* may show atheroma or aneurysm. In *psoas abscess* the psoas muscle is replaced by a thick-walled abscess cavity, which must be followed up to its origin in the bodies of the vertebræ. Clear the bodies of the *vertebræ* and examine them for fracture, dislocation, or disease.

Thorax.—The skin incision and method of clearing the front of the thorax have already been described (p. 196).

Cut through the costal cartilages obliquely downwards and outwards, beginning at the second. The knife is held nearly horizontal, so that when one cartilage is cut through, the haft falls on the next below, and thus the mediastinal contents are protected from injury. If the cartilages are calcified it is better to saw the ribs or divide them with the cutting bone-forceps well beyond the costo-chondral junctions.

Lifting the lower end of the sternum, separate the diaphragm from the costal cartilages, and free the sternum, keeping the knife close to its posterior surface. With the bone-forceps cut through the first rib cartilage, and separate the clavicle at its articulation with the manubrium.

In children the *thymus* gland, lying on the upper surface of the pericardium, behind the upper part of the sternum, and extending up into the thoracic inlet, must be examined, and any morbid appearance noted. Then proceed to examine the *lungs* and *heart*.

The *Pleuræ* are usually opened into in removing the sternum. Note the presence in them of fluid, general or localised, and its nature; exudation of lymph; adhesions, recent or fibrous, and their position. The fluid, as in other serous cavities, may be clear and straw-coloured in hydrothorax; turbid, containing flakes of lymph, in pleurisy; blood-stained in malignant disease of pleura, *e.g.* sarcoma, or in injury to the chest wall or lung. It may be purulent or putrid and offensive in empyema.

Remove and measure the fluid. Normally the pleura contains from half to one ounce of clear straw-coloured fluid, and its surface is smooth and glistening, showing in the adult a varying amount of carbon pigmentation.

Lymph.—On all serous surfaces, so long as lymph can be removed, leaving a smooth and shining, though congested surface, the inflammation is recent. When organisation commences it causes more or less incorporation of the lymphy layer with the surface. Fully organised adhesions are practically non-vascular.

Next open the *Pericardium*. Pinch up a fold of it and incise it from base to apex. From the middle of this incision cut downwards towards the right auricle so as to expose the whole heart.

The contents may be similar to those found in the pleural cavities.

On the epicardium localised, white fibrous thickenings—"milk-spots"—may be present. Tuberculosis and malignant disease of the pericardium are rare and practically always secondary.

The Heart. — Examine the *heart* in situ. Note size, dilatation of individual cavities, and their proportion to each other. In great dilatation of the right auricle the venous sinuses are also distended.

Remove the heart. Lift it by the ventricles well out of the pericardial sac, and cut across successively the inferior vena cava, left and right pulmonary veins, pulmonary artery, aorta, and superior vena cava.

Open the cavities in the order in which the blood flows through them.

Right Auricle. — With the curved probe-pointed bistoury join the venæ cavæ, and from the centre of this incision cut into the appendix. Turn out thrombus, and notice whether it is distinctly adherent in the appendix, indicating ante-mortem formation. Note whether the foramen ovale is patent.

Examine the *tricuspid opening*. This normally, in the adult, admits three fingers. Take the cone diameter if desired. Vegetations are rarely present.

Place the heart on its posterior surface, and make an incision into the right ventricle, half-an-inch from and parallel to the septum ventriculorum. Holding the heart up by the margin of the cut pulmonary artery, allow a stream of water to flow into the artery, to test the competence of the valve. If thrombus be present, remove it before using the water test. If the water be retained the valve is competent. Take the cone diameter if desired. Continue the incision in the ventricle upwards into the pulmonary artery, cutting the latter close to the septum. The incision, if correctly made, ought to pass between two cusps. Note thickening, or, rarely and chiefly in infants, vegetations on the ventricular surfaces of the cusps.

Examine the *Right Ventricle*. Note the consistence, hypertrophy, or atrophy of the muscle. Note whether the sub-epicardial fat is invading the muscle. Note pallor, or a patchy yellowness of the muscle, the latter indicating fatty degeneration. The ventricle may contain ante-mortem thrombus, adherent between the columnæ carneæ, and, passing upwards into the pulmonary artery, in this situation showing the imprints of the sinuses of Valsalva. The wall of the ventricle varies in thickness normally from one-eighth of an inch at the apex, to one-quarter of an inch near the auriculo-ventricular groove.

Left Auricle.—Join the upper right and lower left pulmonary vein, and continue the incision into the auricular appendix.

Note dilatation, presence of ante- or post-mortem thrombus, thickening of the muscle or endocardium. The endocardium normally is thicker than that in the right auricle. Press the finger down into the *mitral* opening, and note presence of stenosis, dilatation, thickening of the cusps, calcareous deposit, recent vegetations. The orifice normally, in the adult, admits two fingers.

Make an incision anteriorly into the *Left Ventricle* parallel to the septum ventriculorum, as in the case of the right ventricle, and in a similar way test the competence of the aortic valve. Take the cone diameters if desired.

Dissect away the *aorta* from the pulmonary artery, and extend the incision in the ventricle upwards into the aorta, passing between two of the cusps.

Examine the aortic cusps for vegetations, thickening, contraction, dilatation, adhesions in the angles, calcification; infective redundant granulations, ulcerating in their centre, destroying the cusps, and extending on to the wall of the aorta or ventricle. Slit up the *coronary arteries*, and note presence of atheroma or calcification in their walls and that of the aorta.

Note thickening and contraction of chordæ tendineæ, accompanying a similar change in the mitral segments, indicating old endocarditis.

Examine the wall for cloudy swelling, fatty degeneration and infiltration, myocarditis, infarct, and aneurysm.

Cloudy swelling causes pallor (often patchy) of the muscle, chiefly near the endocardium. Fatty degeneration and infiltration show appearances similar to those described in the right ventricle. If sections be made across the ventricle, near the apex, small white patches in the midst of the muscle indicate chronic fibroid myocarditis. Hæmorrhagic infarcts appear as brownish necrosed patches in the muscle. Aneurysms are localised dilatations near the apex of the ventricle, and result from myocarditis or infarct.

Note dilatation and hypertrophy of the walls. The ventricle in the adult measures about three inches from base to apex internally. The thickness varies from one-quarter of an inch at the apex to one-half to three-quarters at a short distance from the auriculo-ventricular groove. The papillary muscles and columnæ carneæ usually partake in the hypertrophy. Note presence of an abnormal persistent aperture in the septum ventriculorum. This is placed usually just below the right posterior aortic cusp.

If exact measurements of the orifices be desired the "cones" should be passed in the direction of the blood-stream before slitting them up. The normal cone diameters are:—

Aorta, 0·9 in. to 1·0 in.
Mitral, 1·1 in. to 1·2 in.
Pulmonary artery, 1 in. to 1·1 in.
Tricuspid, 1·4 in. to 1·8 in.

Weigh the heart after examination, as now all blood-clot is removed. In the adult the weight varies from 9 ounces in the female to 11 ounces in the male. Externally it should measure at least 5 inches in length, $3\frac{1}{2}$ inches in breadth, and $2\frac{1}{2}$ inches in thickness.

The Lungs. — On opening the thorax the lungs, if absolutely healthy, would collapse. As a rule, however, they remain more or less voluminous. Collapse may be prevented by the presence of tough mucus in the bronchi, by vesicular emphysema, consolidation (interstitial or exudative), pleural adhesions, or other causes.

Passing the left hand into the left pleura, free any adhesions present, draw the lung forwards out of the thorax, and divide the root from behind. Remove the right lung in a similar fashion.

Weigh the lungs as soon as removed. The weight of the left lung varies from 15 ounces in the female to 21 ounces in the male, and that of the right lung from 17 ounces in the female to 24 ounces in the male.

Examine the surface. Note any deviation from the normal subdivision of the left lung into two, and the right into three lobes. In *emphysema* the parts affected are distended, pale, spongy, and contain little pigment. The condition is practically always present along the anterior borders. In excessive emphysema there may be large bullæ attached to the lung. Areas of *collapse* are dark in colour, non-vesicular, and depressed below the general surface. Firm areas raised above the general surface occur in lobular pneumonia, particularly tubercular, in infarction, and in stonemasons' lung.

In the very young, and in dwellers in the country, the lungs are pinkish in colour; but in all dwellers in towns, and in workers in mines especially, the lungs show a varying degree of black carbon pigmentation. This maps out the lobules on the outer surface, and follows the interlobular septa, vessels, and bronchi on the cut surface. If excessive the condition is called anthracosis, or coal-miners' lung. The walls of the bronchi and vessels are never pigmented.

There may be pits or grooves on the surface, cicatricial in origin. At the bottom of the pit the lung substance may contain a calcareous, whitish nodule, surrounded by fibrous tissue, all that remains of an old tuberculous condition. Cicatricial grooves may also result from contraction of areas of collapse, or are due to new formation of fibrous tissue along the septa and its contraction. This may occur in syphilis and other conditions causing interstitial pneumonia.

Subpleural hæmorrhages occur in cases of asphyxia, mechanical or occurring in the course of other diseases, and also in toxic and chemical poisonings.

Slit up the larger *bronchi* for a short distance. In acute bronchitis they are brightly congested and contain thick tenacious mucus.

Place the left lung on its posterior surface, with the *base* proximal and the root pointing to the operator's left; then with the long "section" knife make one sweeping cut from the lateral surface towards the root, so that the two halves open out on the root as on a hinge. Incise the right lung in a similar fashion, placing it with the *apex* proximal.

Note the colour and consistence of the lung, and the character of the fluid which escapes from the cut surface on pressure.

Normally the colour of the cut surface depends upon the amount of carbon pigment present; it crepitates between the fingers, and a small quantity of blood, frothy through admixture with air, can be squeezed out.

The surface may be pale in anæmic conditions. It may be deeply congested; bright red in acute, dull red in chronic or hypostatic congestions. If, in addition, the lung be friable, pneumonia is present or commencing. In chronic venous congestion the colour is a dull rusty brown, often patchy in distribution, and the surface dry, unless œdema accompanies.

The lung is tougher than normal, so that the fingers cannot readily meet through it, in collapse and in the chronic interstitial pneumonias.

The consistence of the lung may be increased by deposit in its spaces, or by permanent thickening of its substance.

The former condition is present in the *consolidation* of *pneumonia*, and may affect a whole lobe, as in lobar pneumonia, or scattered lobules or groups of lobules, as in lobular pneumonia. A lobule is the subdivision in connection with a terminal bronchus, and it may be recognised in the adult on the pleural surface as a polygonal area outlined by the pigment lying in the lymphatics in the interlobular septa. When consolidated these areas are devoid of air, the vesicles being filled with exudation or with catarrhal cells. In the early stages of pneumonic consolidation the part affected is congested, in the later stages it is paler in colour. In all stages the friability is greatly increased.

In *œdema* of the lung a large quantity of a watery, frothy, blood-stained fluid can be squeezed out on the cut surface. This does not stain the fingers to any extent.

In *capillary bronchitis* and simple broncho-pneumonia (lobular), muco-purulent fluid may be squeezed out of the smaller bronchi.

Demonstrate the condition of the larger bronchi by slitting them up. In *chronic bronchitis* their walls are thickened and they stand up prominently above the cut surface if the lung be not consolidated. In suspected thrombosis or embolism slit up the pulmonary artery, and follow out its main branches. In the former the blood-clot is adherent; in the latter, if recent, it is not adherent, and may be firm and pale in colour.

Lastly, examine the root glands as to carbon pigmentation, tubercular deposit, caseation and softening, or adenitis.

Some of the more common morbid conditions found in the lungs may now be referred to in greater detail.

In *lobar* or *croupous pneumonia* a large area, usually a lobe or more, is consolidated. There is recent pleurisy on the surface. The lung is very voluminous; on section it is airless and dry. In *red* hepatisation it is of a dusky red colour, and the cut surface is indistinctly granular. Friability is extreme. In *grey* hepatisation—the next stage—the colour is a pinkish grey, the cut surface still dry and distinctly granular, the granules being the loosened plugs expressed from the vesicles by the retraction of their walls.

In *suppurating* pneumonia thick, creamy, sticky pus can be scraped from the cut surface.

In *simple broncho-pneumonia* (syn.: lobular or catarrhal pneumonia), as seen typically following measles in the child, the cut surface is flat and mottled. There are minute grey or yellow areas, sometimes branching, corresponding to the bronchioles and their immediately adjacent vesicles, which are filled with catarrhal cells and leucocytes. Between these the lung is congested, and in some cases—*fibrino-catarrhal* pneumonia—is consolidated from exudation of fibrin into the vesicles.

In the adult, *septic broncho-pneumonias* and *hypostatic pneumonia*, which often has a septic character, show larger consolidated areas raised above the surrounding congested and still vesicular or œdematous lung tissue.

The appearance of *tubercular broncho-pneumonia* is readily distinguished. The consolidated area is usually about the size of a pea, and raised above the surface. Several of these may coalesce to form larger areas. They are yellowish, firm, and may show softening in their centres. In the child the whole lung is found studded with these nodules, and no excavation may exist. In the adult, excavation is present at the apex, the broncho-pneumonic tubercles coalescing to form consolidated areas which break down in their centres. All stages of the process may be followed in the same lung.

Old cavities are distinguished by their smooth fibrous walls; recent cavities by their rough irregular walls, formed by caseating tissue.

Acute Miliary Tuberculosis.—Miliary grey tubercles may be scattered uniformly throughout the lung, in which case the bacilli have been disseminated by the blood-vessels; or they may be clustered along the septa, adventitia of

vessels, or bronchi, following the lines of the lymphatics.

In the former there is also generalised miliary tuberculosis throughout the body; in the latter this does not usually exist, and the spread is more a local one.

Hæmorrhagic *infarctions* occur as consolidated areas, full of blood, pyramidal in shape, with the base to the pleura, over which there may be localised pleurisy. If a section be made the occluded vessel of supply can usually be seen near the apex of the infarcted area.

If necessary, the upper air and food passages may now be removed for examination. Extend the median skin incision upwards nearly to the symphysis menti, dissect away the skin on either side, and remove the soft structures left in the superior mediastinum and neck, as far up as the level of the hyoid bone. Then pass a narrow-bladed knife upwards through the floor of the mouth and free the soft parts from the lower jaw-bone all round; or, if it be advisable, for the sake of appearances, to retain the anterior part of the tongue *in situ*, cut it across about its middle. Draw down the loosened parts through the opening made, then cut across the soft palate at its junction with the hard palate, and detach it with the pillars of the fauces from the base of the skull. Continuing to drag gently upon these, free the remaining attachments of the pharynx to the vertebral bodies, and remove all the parts in one mass.

Laying them down on a flat surface, with the posterior surface upwards, slit up the œsophagus and pharynx with scissors; then, in a similar manner, open up the larynx and trachea. Note congestion of the surfaces, ulcerations, diphtheritic exudations, and new-growths.

In the later period of life the cartilages of the larynx and trachea are frequently ossified, sometimes show calcification or necrosis, the last condition being usually of tubercular or syphilitic origin.

Next, turn the parts so that their anterior surface is upwards, examine the thyroid gland chiefly as to atrophy in cretinism or myxœdema, or enlargement in goitre, or new-growth.

The cervical or mediastinal glands may either be examined at this stage or before removal of the cervical structures from the body.

The walls of the thorax and the spinal column are now exposed, and may be examined more particularly.

First, examine the portion of aorta remaining for atheroma, aneurysm, or contraction.

Removing this, note whether fracture or dislocation of the vertebræ exist, and, passing outwards, examine the ribs for fractures or exostoses.

In rickets the costo-chondral junctions are greatly enlarged, forming beaded projections—"rickety rosary"—which are most apparent on the inner surface.

Sewing up the Cavities.—The examination of the abdomen and thorax being now complete, it remains to restore the body, so far as is possible, to its former appearance.

Remove all fluids from the cavities, replace such organs as are not required for further examination, and pack the thoracic inlet with absorbent wool. Replace the sternum, and with a few stitches secure it in position; then carefully sew up the skin incision, so that no fluid can escape. This is best accomplished by using a continuous "out-and-in" suture, passing the needle through the skin always from within, and pulling steadily on the cord so as to keep the stitches tense. The closer the stitches the better the result. They should not be farther apart than half an inch. Tie the last stitch securely so that it cannot possibly slip.

Then remove all traces of blood from the skin surface, and place a layer of absorbent wool along the line of the skin incision, fixing it, if necessary, with a few turns of bandage. This will prevent soiling of the shroud.

Head and Spinal Cord.—Examination of the brain and spinal cord may proceed after the thorax and abdomen have been closed.

Where the *spinal cord* has to be examined, remove it before the brain. This order is of special importance when the course of ascending or descending degenerations has to be investigated.

Turn the body on its face, and, standing on its left side, draw the integuments as far as possible to that side; then, with firm pressure on the knife, and inclining it outwards, cut down on the left side of the vertebral spines, so as to separate with one sweep all the soft structures down to the laminæ from occiput to sacrum. Passing to the other side, and drawing the integuments to that side, through the same skin incision and in the same manner clear the laminæ of the right side from sacrum to occiput. Then saw completely through the laminæ, near the articular processes. If this be properly accomplished the subsequent procedure is easy.

In the lumbar region cut through one of the interspinous ligaments and the corresponding ligamenta subflava; then inserting the closed points of the cutting bone-forceps between the spines, lever up the upper spinous process. The laminæ generally separate easily, and then, grasping the loosened vertebral arch with lion-forceps, or with the left hand protected by a towel, pull upon it, at the same time snipping with the bone-forceps through the laminæ which are not yet divided. The arch of the atlas vertebra is usually very hard and resistant.

Note any fracture of the vertebral arch or hæmorrhage into the surrounding tissues or into the spinal canal. In the dorsal region a layer of protective fat is usually found lying upon the dura.

Grasp the dura at the lower end of the cord with dissecting forceps, and remembering that the cord does not extend beyond the second lumbar vertebra, cut it across well below that point; then free the dura from its attachments right up the canal, and cut across the spinal nerve-roots as far out as possible. While doing this, and lifting the cord from its bed, traction must always be kept up on the dura sufficient to keep the cord straight, any bending or crushing of it being avoided, as this might produce changes simulating disease.

Cut through the dura at its attachments to the margins of the foramen magnum, and, dividing the cord transversely, close to the skull, remove it.

Examine the cut surface. It should be clean cut, not bulging, and the grey matter distinct from the white.

Lay it on a flat surface, and slit up the dura in the middle line before and behind. Note congestion of membranes, thickening of dura, adhesion to the arachnoid, subdural hæmorrhage, tumour-growth, thickening or calcareous deposit in the arachnoid, presence of purulent lymph or tubercular deposits (miliary or caseating).

Tubercular meningitis occurs most commonly in the cervical and lumbar regions; in the former associated with and in most cases extending from a similar condition at the base of the brain. It appears as a fine granularity or mottling of the surface, best seen on the anterior surface. The anterior surface may be distinguished by the large longitudinal vessels running along the middle line, or by the shape of the anterior horn, and the distinct anterior fissure seen on the cut surface.

If the cord is to be examined microscopically, cut it across at frequent intervals, between the origins of the spinal nerves, and place it in 10 per cent formalin solution.

Brain.—Incise the scalp from mastoid to mastoid across the vertex, first separating the hair along the line of incision. Detach the scalp and occipito-frontalis as far forward as the superciliary ridges, and as far backwards as the external occipital protuberance.

Note presence of hæmorrhage, bruising, or suppuration, external to or under the occipito-frontalis, and any injury or disease of the bone.

Carry the knife horizontally round the skull in a line passing just above the superciliary ridges, and passing through the occipital protuberance. Part of the strong fascia over the temporal muscles should be left above this incision.—The reason for this will appear later, when we come to close the head.

Then make a saw-cut through the skull in the same line, taking care not to let the saw pass through its whole thickness. The left hand or the hand of an assistant steadying the skull must be protected by a towel against injury from slipping of the saw.

"To complete the separation of the skull-cap use the mallet and steel chisel, breaking through the remainder of the inner table, unless a fracture of the bones of the skull is suspected, in which case it is better to use the saw more freely, even at the risk of injuring the membranes or the brain" (Woodhead).

Then lever up the calvarium and remove it, leaving the dura intact supporting the brain. This will not always be possible, for example in old people, in chronic alcoholics, or where the dura has become fixed to the skull from old inflammation. In children, also, the dura is usually firmly attached to the bone. In such cases remove it with the calvarium, cutting it through in the line of the saw-cut.

After removing the calvarium examine its inner surface for fracture, erosions, or prominences.

The anterior fontanelle should be closed at the twentieth month. The cranium may be *thinned* in rickets, congenital syphilis, or hydrocephalus. Note the presence of Wormian bones in the lines of the sutures. It may be *thickened* in old age and the various forms of osteitis. Examine the surface of the dura; slit up the longitudinal sinus, and note whether thrombosis, recent or old, simple or purulent, be present. Then incise the dura in the line of the saw-cut, except in the middle line before and behind, and draw the lateral flaps thus made upwards towards the vertex. Examine its inner surface for pachymeningitis hæmorrhagica, or other condition, and then note the condition of the pia-arachnoid and cerebral convolutions. The arachnoid may be milky from old fibrous thickening, or in recent meningitis.

There may be clear, turbid, or purulent effusion in the pia-arachnoid. The convolutions may be flattened, and the sulci shallow where there is distension of the ventricles, abscess, or tumour of the brain. Note whether this is symmetrical. The convolutions may be narrowed and atrophied, and the sulci deep and wide, filled with fluid in old age, in chronic alcoholism, and in certain other diseases (*v.* INSANITY). There may be tubercles in the pia mater, extending chiefly in the neighbourhood of the Rolandic and Sylvian fissures. These must not be confounded with the Pacchionian bodies which are clustered at the vertex close to the margins of the longitudinal fissure.

In septic and other toxic conditions there may be petechial hæmorrhages in the pia-arachnoid—circumscribed, or more often, diffused.

The membranes may be lacerated in cases of compound fracture. In *hernia cerebri* a mushroom-like projection of brain substance exists, from which, frequently, pus can be expressed.

Congestion. — Passive engorgement having disappeared, as the heart has already been removed and the venous blood drained away, active

congestion is noticed as distinct injection of the delicate vessels covering the convolutions.

Removal of the Brain.—Cut across the attachment of the falx cerebri to the crista galli of the ethmoid. Then insinuate the fingers of the left hand underneath the frontal lobes, and lift them up from the orbital plates. Detach the olfactory nerves from the cribriform plates with the handle of the curved probe-pointed bistoury, and then, still raising and supporting the brain, cut across the cranial nerves, the carotid arteries, and the infundibulum as far from the brain as possible. After dividing the fifth nerves, turn the brain to one side, so as to expose the tentorium cerebelli, which is to be cut through from before backwards, close to its margin. Repeat this on the other side. Divide the remaining cranial nerves and the vertebral arteries; then, supporting the brain with the left hand, or calvarium, gently lever up the spinal cord, which, if not already cut across, should be divided as low as possible, and remove the whole, separating it from the dura.

Weigh the brain. In the adult male the average weight is 3 lbs. 1½ oz.; in the female it is about one pound less.

Examine the *base of the skull* for hæmorrhage, intra- or extra-dural; abnormal prominences—osseous, cartilaginous, or fibrous; localised suppurations or adhesions, especially in the neighbourhood of the petrous bones, and note their relationship to internal or middle ear. Note any obvious morbid change in the cranial nerves. Split up the remaining sinuses and note presence of thrombosis, simple or purulent, particularly in the lateral sinuses, where it may be caused by mastoid disease, and may be attended with cerebellar abscess.

In *acromegaly* or *myxœdema* examine carefully the pituitary body.

Remove the dura and examine the base for fractures.

If the orbits or nasal passages and adnexa are to be examined, the portions of the base covering these must be mapped out and removed with the chisel and mallet.

Examination of the Brain. — If accurate localisation of a lesion be desired, the vessels of the brain should be injected with strong formalin solution, and the brain laid in a similar solution for about a week to harden.

In most cases the brain may be examined at once.

In the first place, examine the *base*. Note variation in the arrangement of the basal vessels, and atheroma or calcification of their walls. Look for thickening of the membranes by fibrous tissue, or for the presence of lymph in the membranes veiling the structures at the base.

In *tubercular meningitis* the membranes contain greenish gelatinous lymph, as far forward as and covering the optic chiasma, and in removing the brain from the skull a large amount of turbid fluid escapes from the subarachnoid space. On opening up the Sylvian and longitudinal fissures, tubercles—little grey or yellow points—are seen along the vessels. These may also extend up over the Rolandic areas, and back over the surfaces of the cerebellum, pons, and medulla. They rarely extend up to the vertex. The lymph may block the foramen of Magendie, and the ventricles be dilated—the so-called "acute hydrocephalus."

In *posterior basic meningitis*—which is usually in reality cerebro-spinal—a somewhat similar but yellower effusion is present at the base, extending from the infundibulum backwards over the anterior part of the under surface of the cerebellum, and over the pons, medulla, and spinal cord. The foramen of Magendie is practically always blocked in this condition, and necessarily the ventricles are dilated. "Chronic hydrocephalus" is caused by the blocking of this foramen by a fibrous membrane produced by such a meningitis. The ventricles may be so distended as to reduce the brain to a thin-walled cyst, and the third ventricle projects at the base as a translucent, rounded swelling, stretching the optic chiasma over it.

In general *suppurative arachnoiditis* the whole brain is covered with a layer of pus or purulent lymph, which may be both internal to and external to the arachnoid.

In all cases of lepto-meningitis the arachnoid becomes milky.

Note the presence of hæmorrhages on the surface, laceration or softening of the cortex, presence of scars, for example, over gummata, and localise them accurately.

Superficial *softening* most often occurs in the area supplied by the middle cerebral artery.

In cases of softening, whether internal or cortical, slit up the arteries concerned, and seek for thrombosis or embolism. These are most frequently found at the origin of the Sylvian artery or its first branch, especially on the left side.

Next examine the *interior* of the brain.

Lay it with the base upwards. Remove the cerebellum, pons, and medulla. Make several incisions into the cerebellum, then separate it from the pons by cutting across its peduncles. Note the condition of the fourth ventricle, and make transverse sections of the pons and medulla, leaving the sections connected in front by the membranes.

Turn the brain vertex upwards, and examine by horizontal slices, cutting from within outwards through the hemispheres, and leaving each slice attached to its neighbours by the membranes on the outer surface.

The centrum ovale may be soft and œdematous, or show numerous small points of congestion.

On reaching the level of the corpus callosum make a vertical sagittal incision for half an inch into the white matter about half an inch from

the middle line, and then cut outwards, exposing the lateral ventricles. This dissection may be sufficient to demonstrate the lesion, or subsequent transverse, coronal, sections may be made across the basal ganglia to determine more exactly the relations of a lesion to external or internal capsules or ganglia, and portions of brain may be removed to expose the horns of the lateral ventricles. The fourth ventricle has been examined in connection with the pons.

If the brain has to be preserved for further microscopic examination, it is better to make the incisions in the following manner—that of Déjerine. After removing the cerebellum and pons, separate the hemispheres, and laying each in turn on its outer surface, make one horizontal section just below the level of the corpus callosum, and then to expose the basal ganglia, make two vertical transverse incisions in the lower part of the hemisphere just behind the anterior extremity of the corpus callosum, and in front of its posterior extremity. These incisions expose most of the important structures in which lesions may occur.

In cases of recent hæmorrhage, after noting its extent and relations, the affected part may be placed under a gentle stream of water, and the brain substance washed away. This exposes the vessels, which should be examined for minute swellings—miliary aneurysms, rupture of which may have caused the hæmorrhage.

In old hæmorrhages there may remain a shrunken, brownish mass, or a cyst with pigmented walls containing a thin fluid.

Newly formed masses in the brain may be of the nature of tumours—most commonly glioma or sarcoma—the glioma infiltrating and with ill-defined outline, the sarcoma circumscribed —tubercular caseous nodules, or gummata.

To examine the *middle ear* it is best to remove the whole temporal bone, and examine it by sections either at once or after decalcifying.

After examination of the brain is completed, return what is not required for further investigation, replace the calvarium and secure it in position by stitches through the divided temporal fascia. In an adult, short spikes may be driven into the diploë, in front and behind, attaching the calvarium rigidly to the base of the skull.

Then sew up tightly the flaps of the scalp so that no escape of blood can take place. It is well subsequently to place a layer of cotton-wool along the line of the incision and secure all with a few turns of a bandage.

No trace of blood or discharge must be allowed to remain on the surface.

This concludes the post-mortem examination.

Occasionally the extremities have to be examined, for example, in cases of fracture, of aneurysms, of suppurations in the tissues round the bones or in the joints. In rickets, in congenital syphilis, and in other conditions affecting the development of bone, it is well to expose the head of the humerus, and, sawing it longitudinally, examine the epiphyseal line.

Part II

A. Preservation of Tissues

1. If retention of the natural colour of a tissue or organ be not desired, wash thoroughly in running water and suspend in 50 per cent alcohol or methylated spirit, distending cavities *loosely* with horse-hair or cotton-wool. Change the spirit at the end of a week, and again after a similar period, if it be decolorised. At the end of three or four weeks mount permanently in 75 per cent alcohol or methylated spirit.

Or, place in 5 per cent solution of commercial "formalin," or of its equivalent, a 40 per cent aqueous solution of formic aldehyde. Change if necessary, and mount in a similar solution or in methylated spirit.

A simple mounting fluid for organs which contain fat is—

Formalin, 5 parts.
Glycerine, 30 parts.
Water, 65 parts.

2. If the natural colour is to be preserved, use a method such as the following, adapted from Professor Jores. Prepare the tissue as it is to be finally mounted, wash away excess of blood, and place in the following solution :—

Formalin, 8 ounces (10 per cent).
Sod. sulphate, 700 grains.
Magn. sulphate, 700 grains.
Sod. chloride, 350 grains.
Water to $\frac{1}{2}$ gallon.

Dissolve the salts in the water and add the formalin or its equivalent solution of formic aldehyde. Inject the solution into the vessels in large organs or tumours so that it may reach the central parts, and fill hollow organs.

Retain in this for forty-eight hours.

Place in strong methylated spirit for twenty-four hours, changing if necessary. The spirit restores the blood colour which had been altered in the formalin solution.

Preserve in pure glycerine 2 parts, water 3 parts, changing if necessary. To the final mounting fluid 1 per cent of formalin may be added to prevent growth of fungi, but this is not absolutely necessary.

These directions may be taken as applicable to most tissues and organs. In the case of hollow organs, such as intestine, a much shorter time is necessary, and the action of the spirit should be carefully watched, as it is apt to decolorise the specimen.

With a little practice and patience excellent results are attained.

B. Preparation of Tissues for the Microscope

Necessaries.—Cathcart microtome and accessories ; methylated ether ; mucilage of gum,

B.P.; picro-carmine (Ranvier's); Ehrlich's hæmatoxylin; Farrant's glycerine mounting fluid; eosine in water (0·5 per cent); absolute alcohol; clove oil; Canada balsam dissolved in benzole; two needles in handles. Clean slides and cover-glasses.

Other reagents or apparatus will be mentioned as they are required.

A tissue can be examined under the microscope when a thin layer of it or its elements, penetrable by reflected light, is obtained. Examination of the surface of an object, using direct illumination without the mirror, is seldom had recourse to in pathology.

A thin layer may be secured by scraping the cut surface, or teasing out a small portion of the tissue with needles, but the most common procedure is to cut thin sections, either by freezing or embedding methods, and subsequently to stain them by suitable reagents which accentuate their component parts. Numerous methods of attaining this end have been recommended, but only such as are in common use, and have been thoroughly tested, can be described here. For others the reader is referred to pathological text-books, such as those by Sims Woodhead, Mallory and Wright, or to the *Pathologist's Vade-mecum*, by Lee.

Examination of Scrapings of Fresh Tissues.—This method has now gone out of fashion, but as it gives good results, and as it is easily carried out, requiring no special apparatus, it merits notice.

Make a fresh cut into the tissue or organ, squeeze out excess of blood if necessary, and with the edge of a knife scrape off some of the fluid from its surface. Transfer a little of this "tissue-juice" to a clean slide and mix with a drop or two of fresh Ranvier's picro-carmine. Gently lower a clean cover-glass on to the mixture, and let it sink by its own weight so as to produce a thin transparent layer of the fluid. Allow the stain to act for a few minutes and then examine. Nuclei of cells are stained with the carmine, protoplasm in varying shades of brown and yellow.

If after five or ten minutes the nuclei are not clearly stained, remove excess of fluid from the slide with bibulous paper, and paint a thin layer of vaseline round the cover-glass to retard evaporation. Examine again in an hour. No mounting medium is required, as the preparation is not permanent.

No more recent method gives a better idea of the natural appearance of the component cells of tumours or other tissues.

Examination of Sections of Tissues.—To obtain the best and most permanent results the tissue must be "fixed" before sections are made, although sections sufficiently good for diagnostic purposes may be got by cutting thin sections of fresh tissues with the freezing microtome, floating these on to a slide, staining for five to fifteen minutes in picro-carmine or in logwood, and mounting in Farrant's glycerine medium.

The ordinary stages in the preparation of permanent sections are—1. Fixation; 2. Hardening, embedding, and section-cutting; 3. Staining and mounting of sections. Several alternative methods of carrying these out have to be described:—

1. *Fixation of Tissues.*—A "fixative" is a reagent which penetrates and kills a tissue quickly, preserves the tissue-elements, and particularly the nuclei, in the condition in which they are at the moment when the reagent acts upon them.

For most purposes one may make a choice of four reagents—(*a*) formaldehyde, (*b*) formaldehyde and alcohol, (*c*) alcohol, (*d*) corrosive sublimate.

(*a*) *Formaldehyde,* or formic aldehyde, is sold in a standard solution of 40 per cent strength in water. A proprietary preparation of this strength is on the market, under the name of "Formalin"; it costs more than the other solution, but its action is identical.

A further dilution of 5 to 10 per cent—*i.e.* 2 to 4 per cent of the gas—of this standard solution is used for fixation of tissues, and tissues so fixed may be cut forthwith with the freezing microtome or after embedding.

Small pieces of tissue up to one-sixth of an inch in thickness are sufficiently fixed in three to six hours. The process may be greatly accelerated by gently heating to blood temperature.

Formaldehyde also hardens tissues, making albuminous bodies firm, but not precipitating them.

All tissues save those in which micro-organisms are to be demonstrated may be fixed with this reagent.

(*b*) *Alcohol.*—Except for fat-containing tissues this is the most useful general fixative, and gives specially good results where logwood is used for staining, or where bacteria are to be demonstrated.

Pieces of tissue not thicker than one-sixth of an inch, and of an area not greater than three-quarters of an inch, are placed in large excess of 70 to 80 per cent alcohol for a few hours, and then transferred to 93 to 95 per cent alcohol for at least two days. The tissue should be supported in the upper part of the alcohol by means of cotton-wool. It has the additional advantage of hardening the tissue.

Ordinary methylated spirit is of a strength of 93 to 95 per cent, and acts as well as pure alcohol.

(*c*) *Formaldehyde (Formic Aldehyde) and Alcohol.*—Ten per cent of the solution of formaldehyde or "formalin" is added to absolute alcohol or methylated spirit. Pieces of tissue of the size already mentioned are fixed in this medium

in a few hours, and at the same time are so hardened and dehydrated that they may be passed on very rapidly to paraffin or celloidin. The alcohol dissolves out any fat contained in the tissues.

(*d*) *Corrosive Sublimate.*—The solution is a saturated one (7·5 per cent) in normal salt solution (0·75 per cent sodium chloride).

This reagent is very generally applicable. Nuclei stain well, fat and bacteria are preserved and perfectly fixed, so that they stain well.

Pieces of tissues up to one-half of an inch in thickness, fix throughout in eighteen to twenty-four hours. After fixation they must be left in running water for twenty-four hours to remove excess of the fixative. They are then transferred to gum and sugar, *i.e.* a mixture of 4 parts gum and 3 parts simple syrup, or if the tissue is to be embedded in paraffin or celloidin, the procedure is as follows :—

Pass the tissue through successive strengths of 30, 60, and 90 per cent alcohol or spirit. To these add sufficient tr. iodi. to give a distinct brown colour. The iodine combines with any mercuric chloride left, producing the iodide, which is more readily dissolved out than the perchloride. If perchloride be left in the tissues it appears in the sections as disfiguring black crystals. The tissues show less shrinkage if they are gradually dehydrated, being left in each strength of alcohol for at least twelve hours. From the pure spirit, or 90 per cent alcohol, they are transferred to absolute alcohol to complete dehydration and thereafter the stages are the same for all methods.

After twelve hours transfer from absolute alcohol to an agent such as cedar oil or chloroform, which will mix with alcohol and dissolve paraffin. Leave in cedar oil until transparent, or in chloroform until the tissue sinks, then transfer to paraffin, melting at 46° C., or about 116° F. ; change after two hours into paraffin, with a higher melting-point 51° C., or about 123° F., and leave until all odour of the cedar oil or chloroform has disappeared. To keep the paraffin melted a small water-bath, regulated at about 52° C., or 125° F., is required. The initial cost is not great. After penetration is complete embed in freshly melted 51° C. paraffin in a paper box, or in the ⌐¯¯¯¯¬-shaped adjustable apparatus sold with the microtomes.

Cool rapidly, holding the whole under cold water as soon as the surface of the paraffin has solidified, so that the paraffin may not form large crystals which might injure delicate tissues.

Embedding in Celloidin.—From the absolute alcohol transfer to a mixture of equal parts of alcohol and ether over night, then transfer to a solution of celloidin, in equal parts of alcohol and ether, of the consistence of thin cream. Cork this up and leave for twelve to twenty-four hours. Then transfer to a solution of the consistence of thick syrup for other twelve to twenty-four hours, and mount on a block of wood or cork of suitable size. Place a layer of thick celloidin solution on the block, put the impregnated tissue on this, and paint on a layer of celloidin all round and over it. As this dries apply other layers until the tissue is well supported. As soon as the whole attains some firmness transfer to and keep in 80 per cent methylated spirit, which hardens the celloidin.

2. *Section-cutting.*—(*a*) From gum and sugar, or from 5 to 10 per cent formaldehyde. Use a Cathcart ether-freezing microtome.

Remove excess of fluid from the tissue, place in a drop of mucilage of acacia B.P., on the zinc plate, and apply mucilage all round to support it. Freeze until the whole is of the consistence of cheese, and cut thin sections.

Instructions for using the microtome are furnished with each instrument.

Transfer the sections from the knife with a camel's-hair brush to a bowl of cold, boiled water or normal salt solution, and mount on a clean slide. The advantage of using boiled water is that, as all the air is boiled out of it, no troublesome air-bells form under the section as it floats in the water.

(*b*) Tissues embedded in paraffin.

Blocks prepared in the manner described may be cut with the Cathcart microtome, being fixed on a special carrier by heating it gently, applying the block, and cooling the whole in water, or they may be cut with the Cambridge rocking microtome. In both cases directions are supplied with the instrument.

(*c*) Tissues embedded in celloidin.

These may be washed out in water, soaked with gum and sugar, and cut like tissues embedded in gum and sugar, care being taken to keep the knife moist with 80 per cent spirit. Transfer the sections as cut to weak spirit. In the case of celloidin better sections are obtained if the knife is carried obliquely through the block, hence a long, heavy knife is to be preferred to the razor ordinarily used in the Cathcart microtome.

If mounted on blocks, the blocks are fixed in a special carrier, supplied, if desired, with the Cathcart microtome, or special microtomes adapted for celloidin may be employed.

3. *Staining and Mounting of Sections.*

Sections cut by any of these methods except the paraffin method may be stained immediately on the slide or in a watch-glass.

Paraffin sections are floated out on the surface of boiled water just under the melting-point of the paraffin, and mounted on clean slides which may be albuminised.

To Albuminise Slides.—Take the white of an egg, shake it up thoroughly with nine parts of distilled water, add about 1 per cent of salicylic acid to retard decomposition, and filter.

Paint on a thin layer on the slide, place against a support, with the albumin downwards, to dry; when dry keep in a covered receptacle.

Albuminising is not absolutely necessary if perfectly clean slides be used.

A slide is placed obliquely under the paraffin section floating in the water, and the edge of the latter drawn on to it by means of a needle. The slide is then gently raised out of the water, bearing the section with it. It is dried and kept at room temperature over night, or preferably in a water-bath, the temperature of which is regulated near the melting-point of the paraffin in order to fix the section to the slide.

Before staining, the paraffin must be dissolved out with benzole, turpentine, or xylol, the solvent washed out with strong spirit, and the spirit washed out with water.

Celloidin sections are usually stained without removal of the celloidin.

Staining of Sections

Picro-carmine.—Cover the section with a few drops of Ranvier's picro-carmine and allow to act for a half to one hour. Remove excess with blotting-paper—do not wash—and gently drop some Farrant's glycerine mounting medium on the centre of the section. Lower a cover-glass on to this, as already described in the preparation of a "Scraping of Tissues" (p. 211). The staining gradually improves.

Hæmatoxylin. — While picro-carmine gives very good results, a preference is shown by most workers for a method which will give a more rapid differentiation of tissues, and especially of nuclei. Such a method is that now to be described. It is a little more complex than the former, but with a little practice facility in its employment is readily attained.

As the active colouring agent in most hæmatoxylin stains is the same—hæmatein—it is only necessary for ordinary purposes to give the method of using one of the more dependable of the hæmatoxylin stains—that introduced by Ehrlich.

Ehrlich's hæmatoxylin may be bought ready for use, or may be prepared as follows:—Hæmatoxylin, 2 grains; absolute alcohol, 60 cubic centimetres. Dissolve, then add—glycerine, 60 c.c.; distilled water, 60 c.c.; glacial acetic acid, 3 c.c.; ammonia alum to excess.

Expose to the light, frequently opening and shaking until the liquid acquires a deep port-wine colour. This usually requires two months. If carefully stoppered the staining powers remain constant for years.

Stain sections of "fixed" tissues for fifteen minutes.

Apply a 1 per cent solution of hydrochloric acid in water or spirit to limit the stain to nuclei. This changes the section to a distinct red colour. The hæmatoxylin at this stage is acid and permanent, so that it must be rendered alkaline by washing in tap-water for one to two hours, or for a few seconds in water to which a drop of strong ammonia has been added. After using ammonia wash thoroughly in tap-water.

The hæmatoxylin is now blue and practically permanent.

Counter-stain for one minute in 0·5 per cent solution of eosine in water, wash in water, pass through methylated spirit and absolute alcohol to dehydrate, clarify in clove oil, benzole, turpentine, or xylol, and mount in Canada balsam dissolved in benzole or xylol.

It must again be noted that sections stained in picro-carmine must be mounted in Farrant's medium only.

If picro-carmine sections be dehydrated in alcohol and mounted in balsam, the picric acid is dissolved out wholly. If, however, a trace of picric acid be added to the clarifying agent the colour will be restored, but the result is not very satisfactory except in the hands of skilled workers.

For ordinary purposes of diagnosis no other stains are required.

Fat.—To demonstrate the presence of fat, sections may be cut by any of the freezing methods detailed, and stained black with osmic acid or orange with sudan.

Osmic acid is used in $\frac{1}{5}$ to $\frac{1}{10}$ per cent solution in distilled water, a stock solution of a 1 per cent strength being kept in a black stoppered bottle.

Sudan iii. is employed as a saturated solution in 70 per cent alcohol. In both cases the stain is applied till the required colour is obtained. The osmic acid preparations are mounted in Farrant's medium, or dehydrated and mounted in balsam; the sudan preparations can be mounted in Farrant's medium only.

For the special methods of staining applicable to nerve cells and fibres the reader is referred to Ford Robertson's *Pathology of Mental Diseases.*

C. Bacteriological Investigations

This subdivision of the paper should be read in conjunction with earlier articles on Fluids, Bacteriological Examination of; Micro-organisms; and Microscope.

Bacteria may be examined as they occur in tissues or fluids, or after separation from these by cultivation on suitable media, or by inoculation into animals.

This last expedient need not be referred to further than to impress upon the reader the fact that it may not legally be practised unless in a place specially registered for the purpose, and unless the investigator have obtained a special license under Act 39 and 40 Vict.

The routine method of examination may be summarised thus, taking the case of a pathological fluid:—

1. Examine films stained by "simple" and "differential" methods, using a high power

(700 to 1000 diameters or more). In some cases this may suffice for diagnosis, but in most cases it is insufficient, and the individual organisms are "separated" out by cultivation methods, and then examined in pure cultures, (1) microscopically; (2) by their behaviour in different culture media; and (3), if required, by inoculation into animals. In this manner, sometimes easily, often only after a tedious investigation, the identity of bacteria is made out.

Only methods which require little time in performance, and a minimum of apparatus, shall be described in this article.

A. *Staining* for bacteria, and the deductions to be drawn from the results obtained.

Before staining can proceed thin sections of tissues or thin films of fluids must be obtained.

(*a*) The method of obtaining thin *sections* has been described in the last section of this paper, and here it is only necessary to repeat that tissues are best prepared for bacteriological staining methods by fixation in alcohol or in corrosive sublimate.

(*b*) The preparation of *films* has now to be described.

The cover-glass should be thin, perferably No. 1, for work with oil-immersion objectives (see article on Microscope), and circular, the latter being an important point particularly for those comparatively unaccustomed to work with such fragile articles, as the square cover-glasses are more rapidly broken.

Boil these in 10 per cent solution of hydrate or carbonate of soda; wash thoroughly in tap water; drain and pass through strong mineral or acetic acid; again rinse thoroughly in water, and keep in a covered glass jar in a mixture of equal parts of alcohol and water containing a little pure ammonia.

Be careful not to touch cover-glasses at any stage with the fingers; when prepared in this way, they should be perfectly clean.

Pick up a clean cover-glass with dissecting forceps and wipe dry with a clean, *old* piece of linen or silk, then catch it at the edge with a Cornet's spring forceps.

1. In the case of discharges or fluids generally, make a small loop (one-sixteenth of an inch in diameter) at the end of a platinum needle, *sterilise* the needle by making it glow in a spirit or Bunsen flame; when cool, take up a loopful of the fluid and spread thinly and evenly over the whole surface of the cover-glass, working from the centre to the periphery.

If the discharge be thick, place the drop on one cover-glass and invert another upon it. Allow the fluid to spread out in a thin layer, or, if necessary, exert gentle pressure and *slide* the cover-glasses apart. Place each with the film side uppermost on a piece of white absorbent paper and allow to dry.

2. When making a film from a culture (see later), firstly, place a loopful of water, preferably distilled, on the cover-glass, pick out a minute quantity of the culture with a *straight* sterilised platinum needle, mix up with the water, and then spread out as before.

Drying may be hastened by waving in the air, or by holding the film high over a flame.

Fixation of Film.—Pass the cover-glass, film side up, through a spirit or Bunsen flame three times, at about the rate of the swing of the pendulum of an eight-day clock, pausing at the end of each swing.

The film is now ready for the *stain.*

The colouring agents used belong to the class of anilin dyes, and must be basic. They also act upon cells or tissues, if present, and in order to render the bacteria distinct, these less important objects have to be decolorised. When used in this manner the agent is called a "simple" stain, no other colouring agent being employed.

Under certain circumstances which will be explained later (*vide* Gram's stain, and Ziehl-Neelsen's stain for tubercle), certain organisms retain a stain more firmly than others, so that they resist decolorisation, and stand out very clearly. The decolorised organisms or tissues present may thereafter be stained with another reagent, so as to render the contrast still more distinct. Such a method of staining is termed "differential," and is of great use in determining the identity of organisms.

It will be evident from the foregoing that *over-staining with subsequent decolorisation* is the rule in bacteriology.

It is advisable to obtain the following stock solutions of reagents, with which all the ordinary investigations may be carried out. A few other reagents which are not absolutely essential will be mentioned as occasion for their employment arises.

Stains:—

1. Saturated alcoholic solution of methylene blue.
2. Saturated alcoholic solution of gentian violet.
3. Saturated alcoholic solution of *basic* fuchsin.

Vehicles or Mordants:—

4. One per cent watery solution of caustic potash.
   This is further diluted in compounding the stain.
5. Anilin water.
   Place excess of pure anilin oil in a stoppered bottle; fill with distilled water, shake frequently, and filter twice before using, wetting the filter-papers with water. The water takes up 4 per cent of the oil.
6. Five per cent watery solution of carbolic acid.

Other reagents :—

7. Absolute alcohol.
8. Pure anilin oil.
   This may be used for dehydrating films or sections instead of absolute alcohol, which is very apt to take up water from the atmosphere, particularly when used only at long intervals. The method of employment is described later.
9. Pure sulphuric acid.
   This may be kept in 25 per cent strength.
10. Glacial acetic acid.
    These reagents should be preserved in glass-stoppered bottles.
11. Xylol.
12. Canada balsam in xylol.

Accessories :—

13. Platinum needle in handle. 14. Cornet's forceps. 15. Glass slides. 16. Spirit-lamp or Bunsen. 17. Small cubic-centimetre measure. 18. Filter paper. 19. Six watch-glasses.

When required the reagents are prepared and used thus :—

All compound stains must be filtered immediately before use.

1. Methylene blue. *Lœffler's alkaline methylene blue :—*
   Stock solution of methylene blue, three parts. Aqueous solution of caustic potash (1·10000) ten parts, mix and filter. In the case of the caustic potash, add ninety-nine parts of distilled water to one of the stock solution to make the 1·10000 solution. The made-up stain keeps for about three weeks.
   Method :—
   Stain a film for five minutes; a section for ten minutes. Wash in water. Decolorise in acetic acid, three drops to half a pint of tap-water. Wash again, preferably in distilled water. Dry partially with filter-paper. In the case of a *film*, complete dehydration by holding high over a flame, and mount in balsam forthwith. In the case of a section: Dehydrate rapidly in absolute alcohol. Clarify in xylol. Mount in balsam. *Or :* Remove excess of water with filter-paper. Apply a drop or two of pure anilin oil until perfectly transparent. Wash in xylol and mount as before.

Films may also be dehydrated by these methods.

Risks.—Absolute alcohol is apt to decolorise a methylene blue preparation too much, so that it is well not to remove too much of this stain with the acetic acid.

Alternative Stain.—Thionin blue, 1 gramme in 100 c.c. of a 2½ per cent solution of carbolic acid, may be substituted for alkaline methylene blue, and employed in the same manner. This stain is more permanent and resistant and is preferred to methylene blue by many. Before using, dilute with two parts distilled water.

2. Gentian violet :—
   Stock solution of gentian violet, one part (11 per cent). Anilin water, nine parts (89 per cent). The made-up stain keeps for six to eight weeks. This is a very useful stain, and may be used both as a "simple" and as a "differential" stain. (1) Simple staining with gentian violet. Stain as with methylene blue. Decolorise with methylated spirit or alcohol until of a pale grey colour. Dehydrate and mount in balsam. (2) Differential staining with gentian violet.
   Gram's method :—
   Stain as before. Wash off excess in tap-water. Apply Gram's fixative solution :—Iodine, 1 part (gramme). Potass. iodide. 2 parts (grammes). Water, 300 parts (cub. cent.). For one half minute for films, two minutes for sections. Decolorise in methylated spirit, or 70 per cent alcohol. Contrast stain in 0·5 per cent watery eosine (this is not absolutely essential). Dehydrate and mount in balsam.

It is supposed that the proteid of certain organisms forms with the gentian violet and the iodine a compound insoluble in alcohol.

A modification of this method which sometimes gives better results, particularly in the case of sections, may be described, as it can be carried out with the agents at our disposal. It may be used in all cases in which Gram's method is indicated.

Weigert's method :—
Stain with gentian violet. Wash in 1·600 solution of common salt. Dry with filter-paper. Apply the iodine fixative solution. Dry with filter-paper. Decolorise, and at the same time complete dehydration with anilin oil, three parts, and xylol one part. Wash in xylol and mount.

3. Fuchsin. *Ziehl-Neelsen's method.*—This stain is used especially for demonstration of the tubercle bacillus. Stock solution of fuchsin, 1 part. Five per cent carbolic acid, 8 parts.

Or add the fuchsin solution to the carbolic acid in a test-tube until precipitation begins. On heating, the turbidity disappears.

Filter some of the stain into a watch-glass. Heat gently until all turbidity or surface metallic shimmer disappears, then drop in the film or section, face *down*. Continue heating gently, until a bubble of steam rises, then keep hot for five minutes. Remove the film or section. Wash in tap-water and decolorise thoroughly in spirit or alcohol. Complete decolorisation in 25 per cent sulphuric acid, allowing to act for five minutes. Wash in tap-water. For contrast stain use *alkaline methylene blue*, or aqueous methylene blue, made by adding a few drops of stock solution to distilled water until a deep blue is obtained. Dehydrate, clear, and mount.

It is important to commence decolorisation with alcohol, as this affords a fairly accurate means of differentiating the tubercle bacillus from other "acid-fast" (acid-resisting) organisms.

Spores.—If the above stain be allowed to act longer—fifteen to twenty minutes—it may be used for demonstrating spores in organisms which propagate by that method, weaker sulphuric acid (1-5 per cent) being used to decolorise.

Flagella.—For methods of staining flagella the reader is referred to any of the text-books on practical bacteriology.

There is a considerable number of diseases in which one may hope for help in diagnosis by adopting the above procedures—for example, suppurations, abscesses localised or spreading, gonorrhœa, periostitis and osteomyelitis, erysipelas, pneumonia, tuberculosis particularly pulmonary, leprosy, influenza, anthrax or malignant pustule, acute spreading gangrene, tetanus, plague, actinomycosis.

Although given a list of such favourable length, the reader must not conclude that, provided the staining methods be carried out correctly, he will be able therefrom to formulate a diagnosis in every case he examines, conditions clinically similar being frequently caused by bacteria which are quite dissimilar. Still, in the majority of cases one may expect assistance, and in very many one can make an accurate diagnosis from such examinations.

In abscesses one may expect to find most commonly cocci singly, in clusters, or in chains. The films should be stained with a simple stain, or by Gram's or Weigert's methods. The pyogenic cocci retain the stain by the latter methods. To determine their identity they must be separated by cultivation on suitable media. Chains (streptococci) are usually found in acute, rapidly spreading abscesses.

Suppurations occurring during the course of pneumonia and typhoid, whether of soft parts or bone, may be caused by the organisms associated with these diseases. The diplococcus of pneumonia (Sternberg and Fränkel) may often be readily distinguished by staining methods, its double lanceolate shape and capsule marking it out from other cocci present, especially if stained by Gram's method; but if bacilli are found in abscesses during typhoid fever one must not conclude without very careful investigation, for which great experience and special methods are required, that these are the bacilli of typhoid, as many other organisms may be present. In empyema the diplococcus of pneumonia should be looked for (see p. 151).

In periostitis and osteomyelitis bacteriological investigation of the pus is usually not carried out to diagnose its presence, as that depends more on clinical data, but to determine its character so as to direct treatment. The most common organism found is the staphylococcus pyogenes aureus, but streptococci may be found, and their presence in certain cases may call for the use of anti-streptococcic serum.

Where the suppuration is tuberculous in origin one may frequently by Ziehl-Neelsen's method demonstrate the presence of the tubercle bacillus.

Gonorrhœa.—When a suppurative urethritis with production of creamy greenish yellow pus occurs shortly after connection, one may hope by staining simply with methylene blue or thionin to find gonococci of characteristic double character in the protoplasm of the pus cells. This absolutely confirms the diagnosis, the only organism which might cause confusion—so far as is known—being a diplococcus occurring in puerperal cystitis, and this stains by Gram's method, whereas the gonococcus becomes decolorised. In later stages the bacteriological diagnosis cannot be so confident, and cultivation in Wertheim's medium has to be employed. This is composed of a mixture of human blood and agar. Its method of employment need not be referred to here.

In *erysipelas*, fluid from the vesicles at the spreading edge or obtained by puncture may be examined by staining or cultural methods, and found to contain streptococci.

Pneumonia.—The "rusty" sputum should be examined by "simple" stains or by Gram's method to demonstrate the diplococcus with its capsule.

Pulmonary Tuberculosis (clin. *phthisis pulmonalis*).—The sputum from a suspected case mixed with 1·20 carbolic acid if desired, should be placed in a flat glass dish, and the small yellow points picked out and crushed between cover-glasses, or, if these be not prominent, a drop of the yellow part of the sputum should

be taken. The films are stained by Ziehl-Neelsen's method. The tubercle bacilli stand out as red rods upon a blue ground. Other organisms present are stained by the methylene blue contrast stain.

A scraping from a *leprous* nodule or sections of it may also be stained by this method. The bacilli are found in clusters in the "lepra" cells, and also free.

Influenza.—The bacillus of influenza is very minute and difficult to demonstrate. The best stain to use is Ziehl-Neelsen's, diluted with four times its bulk of distilled water, and filtered. This should be allowed to act for half an hour, and subsequent decolorisation in weak acetic acid should not be carried far. The bacilli often occur in clusters, the long axes pointing mostly in one direction, and they show end-staining, the central part of the rod being only faintly stained, so that they may resemble minute diplo-forms.

Anthrax.—The bacillus is of large size and thick, and may be demonstrated by Gram's method in sections of, or in fluid from, the vesicles on the raised edge of the malignant pustule. Cultivation methods should also be practised.

Acute Spreading Gangrene.—The most important variety of this is malignant œdema, and great care must be employed—as in the case of anthrax—to avoid inoculating one's self. Here again fluid from simple puncture, or obtained in a sterilised hypodermic needle, may be examined for the bacillus of malignant œdema, which may present an oval spore lying near the centre of the rod. This must in addition be examined by anaerobic culture methods.

In cases of *tetanus* the discharge from the primary wound may be examined by staining, but the bacillus is not always found in this way. Cultural methods should always be tried.

Plague. — Fluid obtained by hypodermic puncture is examined by simple staining methods, and by culture on agar. Here again excessive caution is demanded. Buboes, when punctured, usually break down early. In pneumonic plague the sputum may show both plague bacilli and Fränkel's diplococcus, and it may be difficult to distinguish between them, even though the former does not stain by Gram's method. The plague bacillus is usually somewhat oval, and shows marked end-staining.

Actinomycosis.—The pus is thin, and contains minute yellowish points, which should be picked out and examined after staining by Gram's method. Usually an irregular felted collection of filaments is found, in which dichotomous branching may be distinguished.

These are some of the diseases which may be met with by the practitioner, in which bacteriological investigation by staining methods assists diagnosis.

B. Investigation by *cultivation* on suitable media, such as beef-broth, agar-agar, peptone gelatine, glucose agar, and blood serum.

Beef-broth, which is also the basis of agar-agar and peptone gelatine, is made as follows:—

Place in an enamelled pan $\frac{1}{2}$ lb. (500 grammes) of minced *lean* beef. All visible fat must be removed. Add rather more than one litre (35 oz.) of water, and macerate for an hour at a gentle heat considerably below boiling-point, then bring slowly to the boil. Allow to cool so that the fat solidifies, filter through several layers of muslin, and make up to one litre. To this beef-extract, which is simply a solution of the flesh extractives and salts, and is closely analogous to normal urine, is added nutrient material in the form of digested albumin (peptone) and common salt.

Very good beef-extract may be made by dissolving 3 grammes of Liebig's extract of meat in one litre of water.

To make beef-broth (*bouillon*) take: peptone, 10 grammes (1 per cent); common salt, 5 grammes ($\frac{1}{2}$ per cent); beef-extract, 1 litre; or smaller proportionate quantities. Dissolve with the aid of heat. Test the reaction of the result with litmus paper. If acid, render faintly alkaline with a few drops of 10 per cent solution of caustic soda. Again boil thoroughly, and filter through moistened filter-paper into a perfectly clean glass flask or into test-tubes. Plug with unmedicated cotton-wool, and sterilise for an hour on three successive days in a Koch's steriliser or in a potato-steamer.

The broth is then ready for use.

A solid medium is, however, for many purposes more convenient, and to obtain this, agar or gelatine (finest gold medal) is added to the beef-broth. The former is for incubation at high temperatures, the latter for cultivation at room temperatures. The agar and gelatine of themselves have no nutrient value.

The method of preparing a litre of each is described. If properly sterilised, they keep indefinitely.

Agar-gelatine, or shortly "Agar."

Agar-agar, 20 grammes (2 per cent).
Beef-broth, 1 litre.

The agar is cut up in small pieces and soaked for a quarter of an hour in about 1·500 acetic acid in water. It is then collected in a piece of muslin and washed thoroughly in tap-water until all traces of acid reaction disappear. This treatment facilitates solution.

Boil thoroughly in an enamelled pan, avoiding burning, until all the agar is dissolved. See that the reaction is still slightly alkaline; cool down to about 45° C., and add the white of an egg with the shell which have been beaten up with 100 c.c. of water. This water helps to make up for loss by evaporation. Shake up and boil again thoroughly, stirring the while to prevent burning, until the egg coagulates and

separates. Filter *hot* through special agar filter-paper or through pure cotton-wool or "glass" wool. The former gives the better result, and use of a hot water jacketed filter renders the operation easier. Tubes may be filled and plugged at once, or the prepared agar kept in a plugged glass flask, the plug being covered with tin-foil.

Tubes and flask are sterilised as in the case of the broth. During cooling the tubes are inclined so that the agar solidifies with a large surface. Water of condensation collects in the bottom of the tube.

Glucose agar is made by adding 1 to 3 per cent of glucose to agar. The tubes are half-filled, and allowed to cool in the erect position.

Peptone gelatine, or simply "Gelatine."

Finest gold medal gelatine, 100 grammes.
Beef-broth, 1 litre.

Dissolve with *gentle* heat—preferably in a flask in the steamer, neutralise if necessary, clarify and filter. Fill tubes one-third full, and allow to solidify in the erect position.

The manufacture of the media, particularly of agar, presents certain difficulties, and if they be only occasionally required it is better to purchase them in tubes ready for use from scientific instrument dealers.

Blood serum can be very readily and easily prepared by any practitioner, and is useful for many purposes. The blood may be human or from the lower animals (cow or sheep).

Sufficient blood may be expressed from a placenta to make two or three tubes. With precautions as to cleanliness placental blood, or blood of the cow or sheep, is collected in a small beaker or flask, and after the clot contracts—say in ten hours—the serum is pipetted off into sterile tubes. These are plugged and supported in the steamer in an inclined position. The heat is applied very slowly, and not allowed to reach boiling-point, but only about 80° C., so as to coagulate the serum firmly. This requires one and a half to two hours. The tubes are then sterilised for half an hour on three successive days. The writer has found this method of preparation quite satisfactory, and the resultant solidified serum, though nearly opaque, gives quite as good cultural results as that prepared by other methods, and, moreover, no special apparatus is required.

Lœffler's blood serum, on which the diphtheria bacillus grows best, is prepared by adding two parts of separate blood serum to one part of beef-broth, to which 1 per cent glucose has been added. This is solidified as before.

For the preparation of other media the reader is referred to any textbook of bacteriology.

Employment of these Media

Agar (or Blood Serum).—Take three sloped tubes. Singe off the top of the wool plug, passing the lip of the tube through the flame. If the water of condensation be excessive remove the plug and pour it off.

To inoculate, take a loopful of the material with the sterilised and cooled platinum needle, and holding a tube horizontal, remove the plug by grasping it with the flexed, right, little finger, then passing in the platinum needle, avoiding contact with the walls of the tube, mix up the material with the fluid still remaining at the bottom of the tube. Then draw the loop gently back and forwards over the surface of the medium in a zigzag manner, so as to pass once over most of the surface. Replace the plug, and without recharging the needle repeat this process with the second and third tubes. In the second and third tubes the numbers of organisms ought to be so attenuated that separate colonies will develop on incubating at 37° C. for twenty-four hours.

For this, an "incubator," in which the temperature is kept at a uniform temperature of 35° to 37° C., or 98° to 99° F., by means of a suitable regulating apparatus, should be employed, and is essential for reliable work, though some cultures will develop at room temperature or by placing on a mantel-shelf above a fire. Portions of separate colonies thus obtained are to be picked out with the straight needle, and examined by "film" methods (p. 214).

Gelatine.—This medium may be used in the same way as agar, and incubated at room temperature, that is, from 18° to 22° C.

It is more often used to test colonies which have been separated on agar or serum. Pick out a little from the centre of a colony with the straight sterilised needle, stab this down through the centre of the solid gelatine, and incubate at room temperature.

Some organisms cause liquefaction of the gelatine by a peptonising action, *e.g.* staphylococci, anthrax, cholera; others grow in a characteristic manner, forming a heaped-up mass on the surface (Friedlander's diplobacillus), or sending out lateral processes into the medium (anthrax), or otherwise producing a characteristic appearance.

Glucose Agar.—The tube should be one-half full of *freshly prepared* glucose agar.

This medium may be used in attempting to separate anaerobic organisms, other methods being too complicated for performance by any but skilled bacteriologists.

With a long, straight, sterilised platinum needle take a quantity of the discharge or fluid to be examined, and pass the needle deep into the medium. Replace the plug and cover with a tightly fitting rubber cap. Repeat the process in two other tubes.

Incubate at 37° C. for forty-eight hours; then examine the growth which develops by the film method. If organisms containing spores be found, place the tubes in water kept

at a temperature of 80° C. for half an hour. Then reinoculate tubes from each and incubate as before.

The exposure to a temperature of 80° C. should kill all non-sporing bacteria present.

The resultant cultures may contain any sporing, anaerobic organisms, such as tetanus or malignant œdema, which are often found together.

The reader must be warned, however, that these are very difficult to separate.

Blood Serum.—This is especially suitable for the diagnosis of *diphtheria.*

With the sterilised loop remove a little of the membrane from the throat in a suspected case, inoculate successively three serum tubes, and examine films after incubation for twelve to twenty hours. The diphtheria bacilli develop quickly on this medium, and if present, are readily recognised on staining with methylene blue or thionin blue. They also stain by Gram's method.

A dependable diagnosis cannot be made by simply examining a film made direct from the throat.

Postnasal.—Situated behind the nose, *e.g. postnasal catarrh, postnasal adenoids,* etc.

Postnatal.—Occurring after birth, in opposition to *antenatal* (occurring before birth).

Postpartum.—Following labour, *e.g. postpartum hæmorrhage. See* Labour, Post-partum Hæmorrhage.

Postpharyngeal.—Situated behind the pharynx, *e.g. postpharyngeal abscess. See* Pharynx, Retropharyngeal Abscess.

Postscarlatinal.—Following scarlet fever, *e.g. postscarlatinal nephritis. See* Scarlet Fever (*Complications*).

Posture. *See* Labour, Management of (*Posture in Second Stage*); Labour, Precipitate and Prolonged (*Walcher's Position in Contracted Pelvis*); Position.

Postuterine.—Situated behind the uterus, *e.g. postuterine peritonitis, postuterine cellulitis,* etc.

Postzygapophysis.—The inferior or posterior articular process of a vertebra.

Potain's Aspirator. *See* Aspirator, Uses of (*Apparatus*).

Potain's Syndrome.—The association of certain symptoms in cases of dilatation of the stomach, viz. dyspepsia, dilatation of the right ventricle of the heart, and accentuation of the pulmonary sound.

Pot-au-feu.—A variety of soup, resembling Scotch broth. *See* Invalid Feeding (*Soup-making*).

Potash or Potassium. *See also* Angina Pectoris (*Treatment*); Aorta, Aneurysm of (*Treatment*); Dermatitis Traumatica et Venenata (*Potassium Eruptions*); Diet (*Mineral Constituents of Food*); Hæmoglobinuria (*Toxic*); Kidney, Surgical (*Actinomycosis, Treatment*); Œsophagus (*Inflammation, Causes*); Pharmacology; Syphilis; Toxicology; etc.—The metal itself is not used in medicine, but is represented by a large number of salts.

1. Potassii Carbonas.—A white, deliquescent powder, known as salt of tartar, very soluble in water. *Dose*—5-20 grs. As it is very disagreeable to take, and strongly irritant, it is never used in medicine except for the purpose of preparing other salts. From it is prepared *Liquor Potassæ*, a 5·85 per cent solution of potassium hydroxide (KHO) in water. *Dose*—10-30 m. freely diluted. *Potassæ Caustica* (potassium hydroxide) is made by evaporating liquor potassæ and casting the residue in moulds. Caustic potash has been used locally as a caustic to destroy warts, lupus, etc., but as it diffuses rapidly and it is difficult to limit its action it is not very suitable for such purposes. The scraping of an in-growing toe-nail is facilitated by painting on a 40 per cent solution of caustic potash. In dilute solutions it acts as a sedative to the skin and is employed to relieve itching. Liquor potassæ is rather irritating for internal use, but in small doses it has been recommended as a gastric sedative in dyspepsia, and for its alkaline effect on an abnormally acid urine.

2. Potassii Bicarbonas.—Large, opaque, colourless crystals, soluble 1 in 4 of water, made by passing carbonic anhydride through a solution of potassium carbonate. *Dose*—5-30 grs. It is sometimes used in the treatment of stomach conditions with hyperacidity, but sodium bicarbonate is generally preferable. As a diuretic and for the purpose of diminishing the acidity of the urine its place has been largely taken by the acetate and the citrate of potash.

3. Potassii Citras.—A white, granular, deliquescent powder, very soluble in water, made by acting on potassium carbonate with citric acid. *Dose*—10-40 grs. It is the most agreeable to take of all the salts of potash. It interferes little with digestion, and is used principally for its remote effects as a diaphoretic, diuretic, and febrifuge. It is powerfully antiscorbutic, but is not so valuable for this purpose as lemon juice. It is said to be of value in the uric acid diathesis. It increases the bronchial secretion and diminishes its viscidity, and is of service as a mild saline expectorant. It gives good results as a diuretic in chronic Bright's disease, and is also indicated as a diuretic in feverish conditions. It is a very suitable salt to use for diminishing the acidity of the urine, as for the prevention of the formation of uric acid calculi, and in the treatment of eneuresis when this is due to

over-acidity of the urine. Combined with lemon juice or citric acid it forms a useful cooling drink for feverish conditions, especially in children.

4. Potassii Acetas. — Deliquescent white crystals or masses, very soluble in water, made from acetic acid and potassium carbonate. *Dose*—10-60 grs. Its actions and uses are the same as those of the citrate, but it is especially valuable as a diuretic in chronic kidney disease.

5. Potassii Sulphas—Hard, colourless prisms, soluble 1 in 10 of water, which occur native or are obtained by the action of sulphuric acid as a potassium salt. *Dose*—10-40 grs.

6. Potassii Tartras.—A crystalline powder, prepared by neutralising acid potassium tartrate with potassium carbonate. It is very soluble in water. *Dose*—20-60 grs. (diuretic); 2-4 ʒ (purgative).

7. Potassii Tartras Acidus.—Cream of tartar. Obtained by the recrystallisation of the crude tartar deposited during the fermentation of wine. *Dose* — 20-60 grs. (diuretic); 2-8 ʒ (purgative).

These three salts are excellent saline purgatives, causing large watery motions without griping, and are indicated in cases of dropsy and uræmia where it is desired to abstract fluid from the blood. They are also used in weaker solution as mild saline aperients. They are not so widely employed for diuretic purposes as the citrate and acetate. "Imperial Drinks," useful in feverish states, contain potassium tartrate, glusidum, and oil of lemon.

8. Potassii Chloras. — Colourless crystals with a saline taste, soluble 1 in 16 of water, prepared by passing chlorine into water holding lime or magnesia in suspension and treating the liquid with potassium chloride. *Dose*—5-15 grs. *Preparation* — Trochiscus Potassii Chloratis. 3 grains in each. In virtue of its oxidising properties potassium chlorate is given in stomatitis, in pharyngitis, and tonsillitis, either in the form of the lozenge or as a gargle (10 grains to the ounce). It is especially serviceable in the sore mouth arising from treatment by mercury. Its administration during pregnancy is said to prevent miscarriage, and it is therefore indicated in cases of habitual abortion.

9. Potassii Nitras. — Nitre or saltpetre. White striated prisms, soluble 1 in 4 of water. It is sometimes employed as a diuretic, but the citrate and acetate are much preferable. The fumes from burning nitre relieve asthma, and it is a common ingredient of asthma powders. Pieces of blotting-paper soaked in a 20 per cent solution of nitre and dried may be burned and the fumes inhaled by the patient.

10. Potassii Bichromas.—Large orange-red crystals, soluble 1 in 10 of water. *Dose*—$\frac{1}{10}$-$\frac{1}{5}$ gr. in capsule or as a pill with kaolin. It has been recommended for dyspepsia and gastric ulcer.

Potassii Bromidum. *See* Bromum.

Potassii Iodidum. *See* Iodine.

Potassii Oleas is *Sapo Mollis* or soft soap.

Potassii Permanganas. *See* Manganesium.

Potassii Sulphurata. *See* Sulphur.

Potato Cream. *See* Scurvy, Infantile (*Treatment, Diet*).

Potato Culture. — The growing of bacteria on potatoes as a culture medium.

Potatoes. *See* Diet (*Vegetable, Roots and Tubers*); Diabetes Mellitus (*Treatment, Diet*).

Poteriodendron. *See* Physiology, Tissues (*Nerve, Structure and Development*).

Potio or Potion.—A draught or dose of a liquid medicine or mixture.

Potomania.—Delirium tremens (*q.v.*).

Potter's Asthma or Potter's Phthisis.—A form of fibroid phthisis due to the inhalation of dust by the workers in potteries. *See* Lungs, Pneumonokoniosis.

Pottery Works. *See* Trades, Dangerous (*Lead-Poisoning*).

Pott's Aneurysm. — An aneurysmal varix or arterio-venous aneurysm; Bell's aneurysm.

Pott's Disease. *See* Spine, Surgical Affections of (*Spinal Caries*); *see also* Chest, Deformities of (*Deformities due to Pott's Disease*); Osteo-Arthropathies (*in Pott's Disease*).

Pott's Fracture. *See* Ankle-Joint, Region of, Injuries (*Fractures in Vicinity of*); Deformities (*Talipes Valgus after Pott's Fracture*).

Pouch.—A cul-de-sac or pocket, *e.g.* the pouch of Douglas behind the uterus. *See* Generation, Female Organs of (*Ligaments of the Uterus*); Labour, Injuries to the Generative Organs (*Lacerations of the Vagina*); Pelvis, Perineum and Pelvic Floor (*Vaginal Enterocele*).

Poudrette.—A form of artificial manure made by treating night-soil with sulphuric acid.

Pouges-les-Eaux. *See* Balneology (*France, Calcareous Waters*).

Poulet's Forceps.—A form of axis-traction midwifery forceps.

Poultice.—A cataplasm or external appli-

cation to the skin made usually of linseed meal, bread, bran, mustard, or oatmeal moistened with boiling water, and spread upon linen or muslin, for soothing or for counter-irritant purposes.

Poupart's Ligament.—The fascial band stretching from the anterior superior spine of the ilium to the spine of the pubis.

Poupart's Line.—The artificial line drawn perpendicularly upwards from a point in Poupart's ligament midway between the anterior superior iliac spine and the symphysis pubis; it is one of the lines drawn upon the abdomen to mark it off into regions.

Powder.—In Pharmacy a powder (*pulvis*) is a drug (or drugs) in a finely divided form; there are several official pulveres, such as the *pulvis rhei compositus*, the *pulvis jalapæ compositus*, and the *pulvis opii compositus*. *See also* Dover's Powder; James's Powder; etc.

Powers.—The term applied to one of the factors of parturition, viz. the expulsive forces, the other factors being the passages and the products. *See* Labour, Mechanism.

Pozzuoli. *See* Balneology (*Italy, Muriated Alkaline Waters*).

Practitioner. *See* Medicine, Medical Practitioner, Law as to.

Præ-—In compound words *præ-* (Lat. *præ*, before) means before or in front of, *e.g. præputium* (the foreskin). *See also* Pre-.

Præcordia.—The thoracic region in front of the heart; *præcordialgia* is pain referred to this region. *See* Asphyxia (*Treatment, Hot Water to the Præcordia*).

Præcuneus.—The quadrate lobule seen on the mesial surface of the parietal lobe of the cerebral hemisphere.

Prague Method. *See* Labour, Management of (*Podalic Presentations, Delivery of the Head*).

Prairie Itch.—This name has been given to various itching skin diseases (including scabies) believed to be caused or favoured by the fine dust of the prairie.

Pre-—In compound words *pre-* (Lat. *præ*, in front of) means before or in front of in time or place or value; *e.g. pre-albuminuria* (preceding the appearance of albuminuria), *predicrotic* (preceding the dicrotic wavelet of a pulse tracing), *pregenital* (in front of the external genital organs), *presacral* (in front of the sacrum), and *prevesical* (in front of the bladder).

Preagonal.—Occurring just before death, during the agony or death-struggle; leucocytosis frequently occurs at this time (terminal or preagonal leucocytosis).

Preauricular.—Situated in front of the external ear or auricle, *e.g.* a preauricular appendage or branchial fibrochondroma.

Precancerous.—Occurring before the development of the typical signs of cancer, *e.g.* leukoplakia of the tongue.

Precentral.—Situated in front of the central fissure (the fissure of Rolando) of the brain, thus the *precentral line* in cranio-cerebral topography is one corresponding to the inferior and superior precentral sulci which lie in front of the Rolandic fissure.

Precibiculturists.—"Those people who do not increase their food supply by artificial means, either by cultivating the vegetable kingdom or by breeding animals" (H. Campbell in *Brit. Med. Journ.*, ii. for 1905, p. 40); among the precibiculturists are numbered the most primitive peoples now living, viz. the Australian natives, the Fuegians, Esquimaux, Andamanese, Botocudos (of Brazil), Bushmen (of S. Africa), the Forest Pigmies (of Africa), the Veddahs (of Ceylon), the Ainus (of Japan), and the Digger Indians (of California); the Tasmanians (now extinct) were also precibiculturists.

Precipitate.—Performed with great or unusual rapidity, *e.g.* a *precipitate labour*. *See* Labour, Precipitate and Prolonged (*Precipitate Labour, Causes and Treatment*); Medicine, Forensic (*Infanticide, Cause of Death*).

Precipitins.—Substances causing precipitation from a solution, *e.g.* those "substances in immune serums which cause precipitation when added to the fluids or solutions of substances used in immunisation" (*Brit. Med. Journ.*, i. for 1903, p. 655); "precipitins are elaborated by the injection of albuminous body fluids, such as defibrinated blood, into certain animals whose blood serum, in consequence, acquires the property of precipitating, *in vitro*, the albumins against which the adaptation is directed . . . the injection of milk from one animal develops in the serum of the animal treated a precipitin which is specific for the milk used in the adaptation, but for milk of no other species" (*Da Costa*). *See* Physiology, Internal Secretions or Hormones (*Toxic Action and Immunity, Precipitins*).

Precocity.—Early or premature development of mental or bodily functions; also, when, in infectious diseases, symptoms appear sooner after infection than is usual, they are termed "precocious."

Predigesting of Food. *See* Invalid Feeding (*Peptonised Foods*).

Predisposition.—The physical condi-

tion or state of the tissues of the body which renders them prone to be attacked by some disease, microbic or non-microbic.

Preformation. — In embryology the theory of preformation supposes that all the parts of the fully formed organism exist in miniature in the germ; it is opposed to the theory of epigenesis introduced by Harvey and C. F. Wolff.

Prefrontal. — Situated in front of the frontal bone or of the frontal lobe of the brain.

Preganglionic. *See* Physiology, Neuro-Muscular Mechanism (*Spinal Nerves*, vol. vii. p. 550).

Pregnancy.—This subject will be described in the following sections:—

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A. PHYSIOLOGICAL SECTION

Changes in the Maternal Organs

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As soon as an impregnated ovum becomes implanted in the uterine mucosa a series of remarkable changes sets in, not only in the generative organs, but in adjacent and distant structures, and indeed, the whole general economy becomes materially altered. These modifications may be conveniently divided into (1) local; (2) general.

I. Local Changes

The most important of these are necessarily in the uterus itself. At each menstrual epoch changes of hypertrophy and increased vascularity take place, but if impregnation does not occur, a destructive process sets in resulting in

the menstrual discharge. If, on the other hand, conception occurs, the hypertrophic process continues, giving rise to among others remarkable changes in the size, capacity, and form of the uterus. The modifications which take place in the uterine mucosa, resulting in the formation of the deciduæ, have been described.

Changes in the *size*, *weight*, *capacity*, and *form* of the uterus.

The unimpregnated normal uterus measures 3 inches in length, 2 inches in breadth, and 1 inch in thickness. Some authorities state the measurements of length and breadth as rather less than these—$2\frac{3}{4}$ and $1\frac{3}{4}$ inches. At full term of gestation the uterus has so grown that it measures from $12\frac{3}{4}$ to $15\frac{1}{4}$ inches in length, from $9\frac{1}{2}$ to $10\frac{3}{8}$ inches in breadth, and from 9 to $9\frac{3}{4}$ inches antero-posteriorly. The circumference at the level of the Fallopian tubes is about 29 inches.

The weight of the virgin uterus is about $1\frac{1}{4}$ ounces, that of the full term gravid uterus about 28 ounces.

The surface of the unimpregnated uterus measures $5\frac{1}{2}$ square inches, and its capacity is 1 cubic inch. At the end of pregnancy the surface of the organ is 350 square inches, and its cubic capacity is 400 inches, so that the latter has increased over 500 times.

The *form* of the uterus also undergoes varying modifications as pregnancy advances. During the first three months the uterus retains its pyriform shape: the cervix is not much altered, and is still conical. Expansion occurs chiefly laterally, so that the uterus appears as a spherical body placed over a cervix almost normal in shape. Later, the uterus becomes expanded in its lower part, and by the fifth month it presents a more or less spherical form, the largest diameter being the vertical. At full term the uterus is described as ovoid or egg-shaped, the dome being at the fundus, the smaller end below. However, Spiegelberg has pointed out that the shape of the uterus during pregnancy is by no means uniform. Many deviations occur. The form depends on the primary shape of the organ, on the woman's position, on the position of the fœtus, and on the tension inside the uterus.

Changes in the Position of the Uterus.—Owing to increase in weight and size the uterus necessarily undergoes a change of position as gestation advances. It is very generally believed that during the first four weeks of pregnancy the uterus is situated somewhat lower in the pelvis, and some believe that the flattening of the hypogastrium is an important early sign of pregnancy. Tarnier, however, pointed out that this is not constant, and that very early the fundus rises above the level of the pelvic brim, while the cervix is situated at its normal level in the pelvis. At the end of the third month the fundus is a finger's breadth above the pubis; at the end of the fourth this distance has increased to rather more than two inches; at the end of the fifth to four inches; at the end of the sixth month it is on a level with, or a little above, the umbilicus. A month later the fundus is two inches above the umbilicus, and during the eighth and ninth months it continues to rise till its upper limit lies immediately below the ensiform cartilage. The greatest distance of the fundus from the pubis is attained about fourteen days before full time, for during the last two weeks there is commonly a marked descent, owing to the entrance of the lower pole of the uterus into the pelvis.

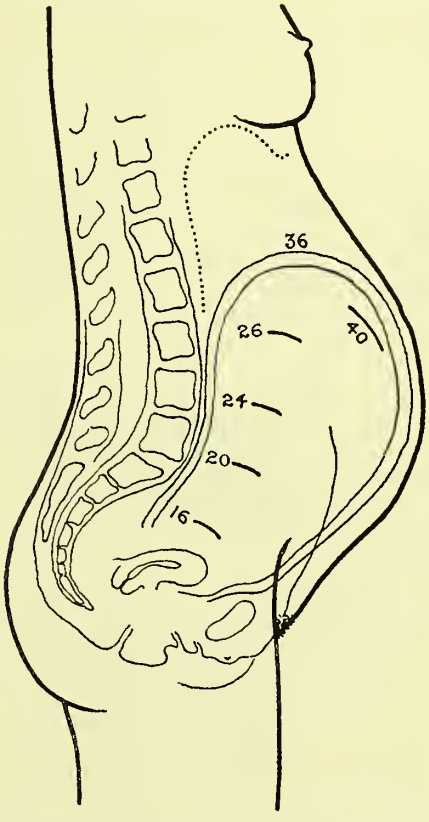


Diagram to illustrate the growth of the uterus during pregnancy.

In multiparæ the abdominal wall is relaxed, so that the uterus projects farther forward, and the fundus does not rise so high as in primiparæ. In the latter, too, the descent of the lower pole occurs earlier. No doubt the rigidity of the uterus itself in primiparæ tends not only to preserve its upward growth, but furthers the early descent of the lower pole.

The situation of the impregnated uterus is normally somewhat oblique, being in the majority of cases slightly rotated, so that the anterior surface looks forwards and to the right, and further, it does not occupy exactly the median position, deviating in most women slightly to the right side.

As to the causes of these deviations there is considerable difference of opinion, and many theories have been advanced to explain them. Some attribute them to the primary *set* of the uterus in the pelvis, some to the fact that women lie most commonly on their right side during sleep, some to the supposed comparative shortness of the right round ligament, some to irregular contractions of the uterine muscle, and others to distension of the rectum.

As gestation progresses the obliquities are maintained, and in the later stages are accentuated. The uterus grows upwards and forwards, so that in the erect position its long axis is in line with the axis of the brim.

In women who have had many children there occurs frequently a much more marked anterior projection of the uterus, and in some extreme cases the fundus is actually dependent.

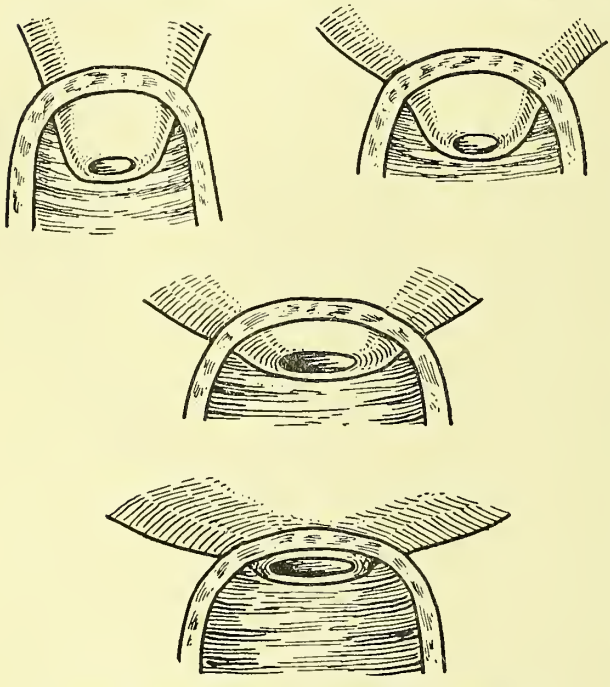


Diagram to illustrate the changes in the cervix during pregnancy.

At the end of pregnancy three-fourths of the anterior surface of the uterus is in close relation with the abdominal wall, though sometimes omentum or small intestines may intervene. The lower fourth is in contact with the posterior wall of the bladder. The posterior surface of the uterus is in contact with the spinal column, and the coils of small intestine find room on either side. The fundus is in contact with the transverse colon and part of the greater curvature of the stomach, the anterior margin of the liver, and the xiphoid cartilage. The ascending colon is in relation with the right border of the uterus, and the descending colon with the left.

Changes in Structure.—At one time it was supposed that the growth of the uterus was the result of mechanical pressure on the part of the enlarging ovum. If such were the case the uterine parietes would be much thinner at full term than in the non-gravid state. As a matter of fact, the thickness at full term is about the same as before pregnancy occurs, and the enormous increase in the size of the uterus is due to hypertrophy of the original muscular wall.

There is, in addition, a distinct alteration in the muscular texture of the uterus, for one of the earliest signs of pregnancy is a softening and yielding of the wall, which strongly contrasts with the dense unresistent pre-gravid character of the muscle.

The thickness of the wall varies in different women and at different parts of the same uterus, for example, at the placental site the muscle is thicker, and in the neighbourhood of the cervix it is thinner than in the virgin state. This hypertrophy is due to an enormous increase in size of the individual muscular fibres. They increase in length from seven to eleven times, and from two to five times in breadth. But besides the normally developed fibres which grow so greatly, there are in the uterine wall embryonic muscle-cells which, when pregnancy occurs, take on active development, and increase to a size commensurate with that of the pre-existing fibres.

At full term the uterus may be divided into three parts: above, the body; below, the cervix; and between these there is developed a portion of the muscular wall, which is much thinner and much less vascular than the body proper. This is known as the lower uterine segment. It is about three or four inches in vertical extent, and is separated from the body by a distinct ridge known as "Bandl's ring," or the "retraction ring." During pregnancy this division has as its lower limit the internal os, but during labour, after the canalisation of the cervix has taken place, the lower uterine segment and the cervical canal are continuous. These parts of the uterus differ both in structure and in function. In the body the muscular fibres are arranged quite irregularly, in the lower segment chiefly vertically, and in the cervix chiefly in a circular manner. The body of the uterus is the only contractile part. The lower uterine segment and the cervix together are expansile.

During pregnancy the cervix uteri undergoes hypertrophic changes similar to those in other parts of the organ. At one time it was believed and taught that from the early months onwards there was a gradual shortening of the cervix, but it is now agreed that this diminution in length is virtual and not real. The apparent shortening is explained by the great softening of the cervical tissue which occurs, and to a

certain extent to the altered direction and position of the cervix. There is, however, a real shortening of the cervical canal during the last fortnight of pregnancy, owing to the gradual dilatation of the canal from above downwards. Even in primiparæ in most cases the os externum is somewhat patulous during the later months, but in some it remains closed till labour sets in. In multiparæ the margins of the os are irregular, and it is always possible to introduce the tip of the finger even before the seventh month.

An examination of the muscular coat of the unimpregnated uterus reveals no definite arrangement of the muscular fibres. Helié has, however, shown that during pregnancy three distinct layers can be differentiated—an external, a middle, and an internal. The external layer, composed of alternating vertical and transverse fibres, covers the body and the fundus of the uterus, commencing at the junction of the cervix in front, and extending rather lower behind. At the fundus the lateral fibres diverge outwards on both sides, and are prolonged along the round ligaments, the Fallopian tubes, the ovaries, and the broad ligaments. The middle layer constitutes the bulk of the uterine wall. The fasciculi which compose it are of variable size, and cross each other in all directions. They surround the veins whose walls consist only of intima, and this arrangement affords an explanation of the arrest of hæmorrhage during the contraction after delivery. The internal layer is composed partly of circular rings of muscular fibres, which are especially well marked round the openings of the Fallopian tubes and round the os internum, and partly of longitudinal fibres arranged in a triangular fashion—one on the anterior and one on the posterior wall of the uterus. The base of the triangle is above and the apex at the os internum. Fibres of these three different layers pass from one stratum to the other, binding all into one continuous muscle.

The muscular coat of the cervix is composed chiefly of transversely circular fibres.

Changes in the Peritoneum and in the Appendages.—Owing to the gradual increase in size of the pregnant uterus the normal relations of the pelvic peritoneum are disturbed. The layers of the broad ligaments are separated; they share in the general hypertrophy, and are carried up by the growing uterus, so that instead of being horizontal they become vertical, or nearly so. As the result of this change of position the Fallopian tubes and ovaries are drawn towards the uterus, till at the end of pregnancy they lie alongside the uterus. The changes in the relations of the peritoneum in front and behind the uterus have not been definitely determined. Pollis's observations led him to believe that the normal level of the peritoneum in the non-pregnant condition was raised during pregnancy both in front and behind the uterus, whereas Webster, from an examination of frozen sections, found the inferior limit of the peritoneal pouches as low during pregnancy as before it.

The round ligaments increase enormously. They become four times their normal thickness, and they so increase in length that at the end of pregnancy they extend from near the umbilicus to the inguinal canal on each side. The utero-sacral ligaments also become enormously developed.

The ovaries are almost doubled in size, and assume nearly a vertical position. Ovulation is in most cases entirely suspended. The Fallopian tubes undergo the usual hypertrophic changes, increasing in thickness and in length. The epithelial lining becomes denuded of its vibratile cilia.

Changes in the Arteries and Veins of the Uterus.—The arteries of the uterus increase in size, in length, and in number. The change in their length is not due to their becoming less tortuous, for Jacquemier has found that they are more tortuous at the end of pregnancy than in the non-pregnant state. The ovarian arteries become about the sixth of an inch in diameter, and the uterine arteries are still larger. The branches on entering the uterus suddenly enlarge, and a very fine anastomosis is established between the two sides of the uterus. Those which pass to the endometrium subdivide freely, and end in a diffuse capillary network. The veins of the uterus become dilated into large sinuses which communicate with each other. The walls of these are intimately united with the surrounding connective tissue, and therefore when the venous channels are mutilated the walls do not collapse, but remain more or less gaping. These veins are devoid of valves, and in the middle muscular layer the wall consists of a single coat. The ovarian veins so hypertrophy that they are almost as large as the external or internal iliac.

Changes in the Lymphatics and Nerves.—The uterine lymphatic system undergoes hypertrophic changes during pregnancy. The whole intra-uterine expanse may be looked upon as one vast lymph sinus (Spiegelberg). The lymphatic channels, when they pass from the substance of the uterus, are collected into a plexus underneath the peritoneum, especially over the fundus and along the sides of the uterus. The lymphatics of the fundus and the body pass to the lumbar glands, while those of the cervix communicate with the pelvic glands. The exact changes in the nerves have not been accurately determined. The cervical ganglion increases in size till it is three or four times as large as in the non-pregnant condition. Some authorities, including Snow, Beck, and Hirschfeld, believed that the nerves remain of the same size as in the unimpregnated uterus.

Robin held that there was an apparent increase of size, but that this was due to increase of neurilemma. Kilian believed the nerve tubes increased in length, but not in thickness; while Schroeder stated that they hypertrophied in every way, just as the lymphatics do.

The Vagina and External Genitals.—Changes of hypertrophy similar to those which occur in the uterus, though to a lesser degree, take place in the vagina and external genitals. The vaginal canal increases in length and in capacity. The submucous adipose tissue becomes less in amount, but there is marked development of the muscular coat and of the connective tissue. The blood-vessels increase in size and in number, and in consequence of the increased vascularity the mucous membrane assumes a bluish, almost purple, colour which by some is regarded as a very important sign of pregnancy. The mucous membrane of the vagina hypertrophies more than the muscular and external coats, with the result that the normal folds are exaggerated, so much so that frequently these protrude through the vulvar orifice as a violet-coloured swelling. The normal secretion of the vagina is increased, in some cases to such an extent that it forms an actual discharge. The cervix generally occupies a position somewhat to the left of the middle line, and it seems shortened owing to the hypertrophy of the mucous lining of the vagina and the ascent of the uterus into the abdomen. The external genitals become hypertrophied and softened, owing to the increase of vascularity and the development of the lymphatics. The vulva, therefore, appears prominent, the labia being swollen, and the veins seem varicose, while the secretion from the sebaceous and sweat glands is much increased. In multiparæ the vulvar orifice gapes, while in primiparæ it appears smaller and retracted.

The Pelvic Joints.—To a certain extent the articulations of the pelvis share in the changes due to pregnancy. Zaglass held that there was a certain, though very slight, amount of movement in the sacro-iliac joints in the non-pregnant woman. During pregnancy, and especially during the last weeks and during labour, there is a marked serous infiltration of the joints and elongation of the ligaments, and in many women distinct movement of the sacro-iliac and pubic joints can be made out during labour. The sacro-coccygeal joint also becomes more movable.

Adjacent Organs.—The other organs in the pelvis undergo changes during pregnancy, partly due to excessive congestion similar to that which occurs in the uterus, and partly due to mechanical pressure. During the early months the pregnant woman suffers from irritability of the bladder, frequent micturition, and sometimes incontinence. These symptoms are due partly to pressure by the growing uterus, and partly to hyperæmia of the vesical mucous membrane. Towards the end of pregnancy these symptoms recur, owing to the sinking of the uterus; and in some cases retention of urine results from the pressure of the presenting part on the urethra.

Nearly all pregnant women suffer from constipation. This, if not due to persistence of habit, is the result of pressure of the gravid uterus upon the sigmoid flexure, interference with the normal peristalsis, and the limited action of the abdominal muscles.

Œdema of the lower extremities and a varicose condition of the veins are very common during pregnancy. These, during the later months, are the result of increased intra-abdominal pressure, while in the early months the same changes may occur merely from the slowing of the blood current consequent on the dilatation of the pelvic veins. Pains in the lower extremities are referred to pressure upon the nerve trunks from the lumbar and sacral plexuses.

The Abdominal Wall.—The distension of the abdominal walls during pregnancy leads to the production of the "striæ gravidarum." These are bluish red lines which become more conspicuous in the later months, and are found over the lower part of the abdomen, and especially the sides. After labour they persist, and gradually become white in colour. These striæ are the result of the stretching of the deep layer of connective tissue of the cutis. During pregnancy these lines are also seen on the nates, the thighs, and the breasts. They are not peculiar to pregnancy, for similar marks may appear on the abdominal wall when it is rapidly stretched, as from a tumour or ascites. Other pigmentary changes in the skin occur, but they are not constant. The most common is the appearance of what is called by some the "linea nigra," a dark-brown line which runs from the symphysis pubis to the umbilicus, or even beyond it. Patches of discoloured skin sometimes appear on the face, especially on the forehead; but these pigmentary changes in the skin are of no diagnostic value in pregnancy.

By the fifth month of pregnancy the umbilicus commences to be less deep, by the seventh the depression is obliterated, and from this time onwards it protrudes more and more till it forms a rounded elevation on the abdominal wall.

During the last three months of pregnancy a physiological separation of the recti muscles occurs in the great majority of cases. The elasticity of the abdominal muscles is lessened, and when the distension is very great an actual loss of tonus takes place. The muscles which chiefly suffer in this respect are the transversalis, the external and internal oblique muscles; while the recti, on account of their length and thickness, suffer to a much less extent, if at all.

Changes in the Mammæ.—From the second

month onwards hypertrophic and other changes take place in the mammæ, resulting in gradual increase of not only glandular tissue, but of fat and connective tissue. The periphery of the breast is first affected. It becomes irregularly hard, and presents knotty masses which are enlarged acini of the glandular structures. Later similar hypertrophic changes spread along the ducts of the gland, towards the nipple, till ultimately the whole organ is affected. The distension of the skin leads to the formation of striæ similar to those seen on the abdominal wall, and through the tightly drawn skin a network of veins can be seen, especially at the margins of the organs. The nipple becomes enlarged, more sensitive, and more readily erectile, and the pink areola surrounding it is replaced by an areola which varies in colour according to the complexion of the woman. In fair women it is of slight brownish colour, whereas in brunettes it is dark brown, or almost black. At the end of two months the areola is slightly raised, and the sebaceous follicles become more and more enlarged till they form distinct tubercles on the surface of the darkened area. These are known as Montgomery's tubercles. The usual diameter of the mammary areola is from one to one and a half inches; but in many cases, from the fifth or sixth month onwards, a "secondary areola" surrounding the primary is formed. This is not so deeply coloured, and is not always very marked. From the third month pressure on the gland causes the exudation of a thin fluid known as the colostrum; it increases in quantity as the gestation advances, and is the product of the newly formed and of the developed pre-existing gland cells. It is an albuminous fluid, and contains fat cells, epithelial cells, and typical "colostrum corpuscles."

II. General Changes

Pregnancy creates important changes in the different systems and organs of the body. The blood and the nervous system are affected to the greatest extent, but practically all the functions of the body are more or less modified. Since the pregnant woman has to perform nutritive and excretory functions for herself and her fœtus, extensive changes in the general system must occur. So long as these changes take place within certain limits health is preserved and improved, but when carried to excess they lead to serious complications.

The Toxæmia of Pregnancy.—Much has of late been written on this subject. It is now believed by some authorities that many of the hitherto supposed physiological symptoms of pregnancy are the result of slight toxæmia, and that the graver complications of pregnancy, labour, and the puerperium are due to intense toxic infection. Vomiting, salivation, pigmentary changes in the skin, neuralgia, irritability of temper, etc., have generally been supposed to be normal since they are so common, but possibly they are due to the presence of toxins. As pregnancy advances, the general economy becomes adapted to the new conditions, and the symptoms of poisoning pass off. The emunctory organs work at higher pressure, but if one of them gives way, then symptoms of toxæmia become intensified, and may become so serious as to endanger the patient's life. Eclampsia is the most striking example of severe toxic infection. It is no longer regarded as a disease primarily of the kidneys. Post-mortem examination reveals important changes in the liver similar to those produced by certain poisons. In many cases of eclampsia no pathological changes have been found in the kidneys, and the clinical evidence of disturbance of the renal function rapidly disappears after labour. The causes of these toxæmic conditions during pregnancy, labour, and the puerperium are varied, and include heredity, climate, previous renal disease, dyspepsia, hepatic disorders, anæmia, and tight lacing. Early recognition of toxic symptoms is important, as active treatment is frequently necessary to prevent serious complications.

Changes in the Blood and Circulation.—At one time it was supposed that the chief change in the blood during pregnancy was one of "plethora" of all its constituents, and "bleeding" was a very common operation during the later months of pregnancy. Now it is recognised that though the actual quantity of blood in the body is increased, the change in the nature of the blood is towards anæmia and hydræmia. The watery elements, the white blood corpuscles, and the fibrin are increased; but, on the other hand, the amount of albumin and of red blood corpuscles is diminished. The water increases from 791·1 per 1000, which is the normal amount in the non-pregnant condition, to 819·9 in the last month of utero-gestation. The red blood corpuscles diminish from 126 to 104·49. The albumin lessens from 70·5 to 66·1. The fibrin, which in the non-gravid state is 3 to 1000, lessens until the sixth month; then it begins to increase until, at the end of the ninth month, it is 4·3. The iron in the blood is necessarily diminished in amount, since the number of red cells is so decreased. There are, of course, great variations in individual cases. The hygienic surroundings and the nature of the food of the pregnant woman will materially affect the blood changes.

In 1828 Larcher first pointed out that a physiological hypertrophy of the heart took place during pregnancy. This increase in size is limited to the left ventricle and is such that the whole weight of the heart increases one-fifth during gestation. The increase in the total amount of blood in the body, the placental

circulation, and the amount of abdominal pressure, all involve increased cardiac action, and this shows itself not in more rapid action of the organ, but in hypertrophy and in increased arterial tension. There is also during pregnancy a tendency to thrombotic and varicose condition of the veins. Durozier states that the heart continues enlarged during lactation, but if a woman does not nurse her child it diminishes in size immediately after labour. Women who have borne many children often have a permanent enlargement of the heart.

The liver is also enlarged, and in women who have died suddenly after labour fatty degeneration has frequently been found. The *spleen* shows signs of marked hypertrophy, which is easily understood when one considers its important relation to the quantitative and qualitative changes in blood. It is increased in size from 140 grms. to 180 grms. The *thyroid* gland participates in the general change of increased growth, and is almost constantly somewhat enlarged during gestation.

Changes in Respiration.—Increase in size of the uterus mechanically interferes with the function of respiration. The long axis of the thorax is diminished by the upward displacement of the diaphragm, and this in the later months causes some embarrassment to breathing, but to a certain extent there is compensation in the fact that the lower thorax is increased in breadth. During the last two weeks of pregnancy breathing becomes easier owing to the "sinking" of the uterus and its contents. In the early months a certain amount of dyspnœa is occasionally observed, but this is merely one of the numerous nervous reflex phenomena which so often complicate pregnancy. Since there is more blood in the body during gestation there is naturally an increase of the elimination of carbonic acid gas.

Changes in the Digestive System and in Nutrition.—In the early months of pregnancy the appetite is generally fitful, while nausea and vomiting within certain limits are so frequent as to be regarded as physiological. The varieties, causes, and significance of morning sickness are described under "The Diagnosis of Pregnancy" (p. 230). The appetite of the pregnant woman, even though often capricious to the very end of gestation, generally increases so that the general nutrition is improved. An increase in weight takes place irrespective of the enlargement of the uterus and the presence of a fœtus. On an average the gain is from ten to fifteen pounds, or about one-thirteenth of the whole weight of the body. Excluding the generative organs the adipose tissue is increased more than any other tissue of the body. The deposits are specially marked in the mammary glands, in the abdominal walls, in the hips, and in the omentum. The whole figure therefore becomes fuller and rounder.

The *temperature* of the body is not materially changed during pregnancy, although, according to some authorities, it is slightly lower in the morning than during the day.

Changes in Bone.—Besides the alterations in the bones and joints of the pelvis already referred to, changes in the bones of the skull have frequently been observed in women who have died during parturition. Rokitansky pointed out that irregular bony deposits were found on the under surface of the skull between the bone and the dura mater, and more especially developed on the parietal and frontal bones. These osteophytes consist of calcium carbonate, traces of phosphates, and organic matter. They have been discovered in rather more than one-half of all the cases examined of women dying after the fifth month. Some authorities consider that they are by no means peculiar to pregnancy, so that whether they are physiological or pathological has not been accurately determined.

During pregnancy there is always a risk of delayed union in fractured bones.

Owing to the growth and inclination forward of the growing uterus the centre of gravity is altered so that the pregnant woman is obliged to throw her head and shoulders backwards in order to preserve her equilibrium in the erect posture. The curves of the spine are increased, and this tends to an alteration in gait, which is especially marked in short women.

Changes in the Skin.—The pigmentation of the skin has already been referred to, but other changes are constant phenomena. The sebaceous and sweat glands acquire increased activity. Further, according to Barnes, "women who had been losing their hair when not pregnant found its growth restored during pregnancy, and falling out again after labour." According to some authorities, the changes of pigmentation are associated with a temporary hypertrophy of the suprarenal capsules.

Changes in the Nervous System.—During pregnancy the nervous system becomes especially sensitive, and hence numerous reflex phenomena are observed. Rigors, flushings, fainting, hysterical disorders, disturbance of the special senses, and neuralgia are frequent. In the majority the mental state is one of despondency, especially in the early months, but often as gestation advances towards completion this state gives place to one of "buoyancy and a sense of joyful expectation." It is quite the exception to meet with women who feel unusually well and cheerful throughout the whole pregnancy. Most look forward with great anxiety to labour, fearing the worst for themselves and for their child. Changes in disposition are frequently very marked. A woman who is naturally amiable and bright may during pregnancy become moody and disagreeable, and the reverse is also sometimes observed.

Changes in the Urine.—The kidneys are somewhat enlarged and the urine is increased in quantity, but only in its watery elements. With the exception of the chlorides the solid constituents become less and less in amount as pregnancy advances. The phosphates and sulphates are supposed to be diminished owing to their being used in the growth of the fœtus. At one time it was held that a white, grumous, soft "pellicle," known as kiestein, was diagnostic of pregnancy. It forms in the urine about thirty-six hours after it is passed, and about the fifth day it breaks up and falls to the bottom of the glass; but it is now known that a similar substance is sometimes found in the urine of the non-pregnant and even in the urine of men.

During the last few weeks of pregnancy sugar may sometimes be detected in the urine, and during lactation it may be found in large quantities, diminishing and ultimately disappearing when the milk disappears from the mammæ. This temporary glycosuria must not be confounded with true diabetes, which is a serious complication of pregnancy.

Authorities differ as to the frequency of albuminuria during pregnancy. Schroeder maintains that in all pregnant women the urine contains albumin varying in amount from 3 to 5 per cent; others give the percentage as high as 20 or 30. No doubt albumin is detected in the urine of a very large number of pregnant women, and to many of these the albuminuria is physiological and transitory. This "temporary albuminuria" is much more marked during labour than during pregnancy, and the longer the labour the more likelihood of albuminuria. It is probably due to reflex vaso-motor spasm of the renal arteries resulting from uterine contractions.

The definite causes of albuminuria at any stage of pregnancy are really very uncertain, although many theories have been advanced to account for the condition. Increased intra-abdominal pressure, resulting in interference with the circulation in the vena cava and its branches, has been supposed to account for some cases, but this has never been proved. Most admit that the mechanical pressure of the gravid uterus on the renal vessels predisposes to the disease, and there are several facts which tend to uphold this theory. Albuminuria is more common in the later months of pregnancy than during the first five months. Those cases occur among primiparæ in whom the abdominal walls are tense and unyielding. Further, albuminuria is common in twin pregnancies, and when pregnancy is complicated by the presence of a fibroid or ovarian tumour. It is less frequent during pregnancy than during labour, when pressure is greater, and it diminishes after the birth of the child. The theory of increased intra-abdominal pressure is the oldest of all, but that this is sufficient to *excite* the disease in kidneys previously healthy has never been proved. It has been suggested that the albuminuria which sometimes occurs in the *early* months of pregnancy before there is any possible stagnation from pressure is purely reflex. It corresponds to the reflex irritation of the stomach which results in morning sickness. The hydræmic state of the blood is supposed to account for some cases of albuminuria, and in others the increased arterial tension may be the exciting factor. There is no doubt that in still others the albumin found in the urine of pregnant women is simply the result of vesical catarrh.

It is not necessary to do more than mention these cases in which pregnancy occurs in women suffering from chronic Bright's disease with albuminuria, and those who having previously suffered from kidney disease show albumin in the urine during pregnancy. These cases have quite a different relation to pregnancy from those already referred to.

The presence of albumin in the urine of a pregnant woman is always a cause of anxiety. If it is persistent and abundant, and especially if it occurs early in pregnancy, the outlook is grave, and careful treatment to prevent more serious complications is urgently required. Clinically, albuminuria suggests the possibility of uræmia and eclampsia.

Diagnosis of Pregnancy

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The changes produced in the maternal organism by the condition of pregnancy are so very definite, and opportunities for observing them are presented so often to every medical man, that it might seem impossible for any one exercising due care to make a mistake when he is called upon to decide as to the presence or absence of pregnancy in a given case. That mistakes, however, are frequently made in this connection is a matter of common experience. Such mistakes are doubtless most often due to carelessness on the part of the practitioner—a too hasty examination of the patient, or a too hasty conclusion drawn from subjective phenomena only, without any physical examination. On the other hand, when it is remembered that Tardieu has said (and our knowledge has not advanced much since then) that except the bruit of the fœtal heart, the signs of pregnancy,

from the development of the abdomen and breasts up to the movement and efforts of labour, may be observed when there is no pregnancy, it will be seen that, even by the most skilful and careful observers, errors in diagnosis may occasionally be made.

The ridicule to which the author of such a mistake is apt to be subjected, and the loss of professional reputation which may result, would alone render this subject one of the utmost importance to every one engaged in the practice of medicine. There is, however, another and weightier reason. A wrong diagnosis may imperil the life of the patient. The treatment of certain diseases is very materially modified should pregnancy he also present. In the case of an ovarian tumour, which has been mistaken for a gravid uterus, complications may arise which make its removal both difficult and dangerous; on the other hand, a pregnant uterus which has been diagnosed as an ovarian tumour may be subjected to operation with fatal results, as has unfortunately not infrequently happened.

Whenever, therefore, the question of pregnancy arises, after the history of the case has been carefully searched, a thorough physical examination of the patient should be made. Mistakes will then be rare. It is also a good rule to follow that, however strong the presumptive evidences of pregnancy may be, in the absence of one or more of the certain signs no absolutely definite opinion should be *expressed*. The lapse of a month, while it can but very rarely do harm, will generally be sufficient to clear up the diagnosis.

Pregnancy is revealed by certain symptoms and signs, subjective and objective phenomena. The importance of the latter over the former need hardly be referred to. A patient may not only be mistaken with regard to her sensations, but may purposely make erroneous statements about them. To the physician, therefore, the subjective phenomena afford at the most presumptive evidence only: the certain and infallible signs are perceived by his own senses.

Symptoms. — *Subjective Phenomena of Pregnancy.* — I. *Suppression of Menses.* — Menstruation as a rule is in abeyance during the whole of pregnancy, and its stoppage is usually the first indication to the patient that she has conceived. So constant is this symptom that it forms the most reliable, and therefore generally adopted, datum from which to calculate the onset of labour. It must, however, be remembered: Firstly, pregnancy can occur before puberty, and also after the menopause. Secondly, menstruation may continue during the early months of gestation. Only in very rare cases is it met with after the third month, and most commonly it will occur during the first month only. In these cases the amount of blood lost is generally much less than the patient's normal. This, combined with the short duration of the flow, is often sufficient to arouse suspicion. Cases have, however, been recorded where menstruation went on regularly and normally all through the nine months of gestation, and Montgomery and Palmer record cases where it was never present except during pregnancy. Thirdly, that many other conditions—local, constitutional, climatic, and emotional—may cause amenorrhœa. In this connection the emotional or psychical causes of amenorrhœa call for special note.

Stoppage of menstruation is not infrequently met with in newly married women who are not pregnant but who are very desirous of becoming so, and who anxiously look forward to a failure of the menstrual flow as the first intimation that conception has occurred. More often it will be found in another class—generally unmarried women who have exposed themselves to the risk of impregnation, and are dreading the result. It is in these two classes of cases that mistakes in diagnosis are most easily made, because not infrequently when one such symptom appears, others, also psychical in their cause, are apt to follow, such as morning sickness and swelling of the breasts. To sum up, it may be stated that amenorrhœa, occurring in a healthy woman who has been previously regular, and who has been exposed to the chance of impregnation, is strong presumptive evidence of pregnancy, even in the early months; and its value increases, of course, the longer it continues. Confirmatory evidence must always be sought for, in the early months in the condition of the breasts, and in the later months in the presence of the abdominal tumour. An abdominal tumour associated with amenorrhœa, and whose size corresponds to the number of periods passed, should in every case excite the suspicion of pregnancy.

II. *Morning Sickness.*—Nausea and vomiting are an almost constant symptom in early pregnancy. It occurs, as a rule, in the morning on first rising from the recumbent posture, and hence is familiarly known as morning sickness. In some cases, however, it may occur only in the afternoon or evening. This sickness is generally most severe in primiparæ and in highly strung nervous women. It may come on immediately after conception, more often shows itself for the first time after the first menstrual suppression, and generally ceases during the fourth month. There may be merely a feeling of nausea, but this is usually accompanied by the vomiting of a glairy mucus. Lasting in most cases only a short time during the day, it causes, as a rule, no impairment of health. In some cases, when it becomes exaggerated and no food is retained, it may constitute a very serious danger (*see* Pregnancy, Affections and Complications (*Digestive Disturbances*)). The vomiting of pregnancy is generally considered to be a reflex disorder reflected from the uterus, and from its

occurrence so very early in some cases this view is probably correct. The severer forms of the disorder may, however, be a manifestation of a toxæmia dependent in some way, not yet ascertained, on the pregnant state.

Taken by itself morning sickness is not a symptom of much value in the diagnosis of pregnancy, as vomiting in the morning after rising is not infrequently met with in ordinary gastric catarrh and in chlorosis. When it is associated with amenorrhœa and an unimpaired appetite it is significant presumptive evidence. It is valuable as a corroborative sign because it occurs so early in pregnancy.

Increased salivation is often found accompanying the severe forms of morning sickness. It may be present, however, alone. It is so rarely met with, however, as to be of very slight diagnostic importance.

III. *Various other nervous phenomena* which are occasionally met with in pregnancy—such as abnormal cravings for certain articles of food, changes in temperament (irritability or despondency), and neuralgias—are of no value in themselves as indicating pregnancy. When, however, any of them have been noticed in a patient in a former pregnancy they possess a certain significance.

IV. *Quickening.*—Much more important is the subjective phenomena of quickening, a term used to designate the sensation for the first time by the mother of the movements of the fœtus *in utero*. The date of this is generally about mid-term, when the growing uterus comes in contact with the abdominal wall. As a sign of pregnancy it is obviously of much greater value in multiparous than primiparous women. The movement of flatus in the intestines and irregular contractions of the abdominal muscles may be mistaken even by multiparæ for quickening. Statements regarding the sensation of quickening must, therefore, be accepted with caution, and too great reliance must not be placed on them. If the fœtal movements have not been felt by the mother this must not be taken as proof of the non-existence of pregnancy, for they are sometimes not noticed until very late, sometimes not at all even when the child is quite healthy; and, lastly, they will be absent, of course, when the fœtus is dead.

V. *Bladder Irritation.* — Frequently during the first three months, while the uterus is still a pelvic organ, patients complain of some bladder irritation. Generally this is evidenced by frequent calls to micturition, in some cases by actual pain during the act. This is due to the pressure of the anteflexed uterus against the bladder, and passes off as soon as the uterus rises out of the pelvis. By itself this symptom is, of course, of no value, and even along with others is of little importance in diagnosis.

Kiestein, the flocculent deposit sometimes present on the surface of the urine in pregnancy, has no diagnostic significance as was once supposed. It is composed of fungi mixed with triple phosphates. It is found also in the urine of non-pregnant women and also in that of males.

The Objective Signs of Pregnancy.—These, as already stated, are of much greater diagnostic value than the subjective symptoms just mentioned. These signs are made known to us by direct physical examination of the patient. This includes an examination of the mammæ, abdomen, vagina, and uterus.

Changes in the Mammæ.—These have already been considered under the head of local changes in the maternal organism resulting from pregnancy. We need, therefore, refer only to their relative value in diagnosis.

Montgomery regarded these changes to be *certain* signs of pregnancy if they were well marked. As a general rule this may be taken to be true if they are found present in a woman who is known not to have been previously pregnant. In parous women, however, their value is much diminished by the fact that in them the darkening of the areola, the prominence of the areolar tubercles, and secretion may be found present for years after lactation has ceased. They have been found present also in non-pregnant girls, it is stated, as a result of repeated sexual intercourse and mechanical irritation of the breasts and genitals. Almost identical changes, too, it must be kept in mind, are found occasionally in connection with various pelvic disorders, such as ovarian and fibroid tumours, chronic metritis, etc. The chief value of the mammary changes to the physician lies in the fact that they occur so early, and also because they can be so easily examined in a suspected case of pregnancy without exciting any suspicion in the mind of the patient as to the reason of the examination.

Abdominal Signs of Pregnancy. — The changes in the size, shape, and appearance of the abdomen have already been stated. There are, however, other and more reliable signs. They are as follows:—

(*a*) *Active Movements of the Fœtus.*—These can generally be both seen and felt through the abdominal wall after the sixth month. In a multiparous patient they may be felt even much earlier. As seen these active movements must be differentiated from intestinal peristaltic movements. When felt in abdominal palpation they can hardly be mistaken for anything else.

(*b*) *Passive Movements of the Fœtus, Ballottement.*—The sensation which is imparted to the examining hand when the fœtus is moved about in the liquor amnii is called ballottement. It can be felt on abdominal examination if the fœtus is not too large, or the liquor amnii not too scanty, and is therefore most easily recognised about the sixth month. It is, however,

subject to certain fallacies. A solid part of an ovarian tumour, when moved between the two hands, may impart a sensation similar to that of the fœtus in the liquor amnii. As practised in vaginal examination, when the patient's shoulders are raised, ballottement is one of the most reliable signs of pregnancy that we possess. It can generally be felt from the fifth to the eighth month, and can be simulated by nothing else. Vaginal ballottement is of special value in the diagnosis of hydramnios, when many of the ordinary signs of pregnancy are observed. It these cases vaginal ballottement is most clearly made out. It is of value also because it can be practised when the fœtus is dead if the liquor amnii has not escaped. It should be noted also that not only may a large fœtus and absence or deficiency of the liquor amnii obscure this sign, but that a malpresentation, such as a breech or placenta prævia, may make its recognition difficult or impossible.

Intermittent Contractions of Uterus. — The consistence of the uterus during pregnancy as felt on abdominal or vaginal examination will be found to vary from time to time. This variation is due to intermittent contraction and relaxation of its muscular wall. The interval between the contractions varies from five to fifteen minutes, and the duration of a contraction is from three to five minutes. On bimanual examination they can be felt as early as the third month, and they continue throughout pregnancy. They are not absolutely positive signs of pregnancy, as they are occasionally felt in soft, rapidly growing fibrous tumours, but so very rarely that their presence may be taken as very strong presumptive evidence. They continue though the fœtus is dead.

Fœtal Heart Sounds.—Mayor of Geneva in 1816 first pointed out that on auscultating the abdomen of a pregnant woman after mid-term the sounds of the fœtal heart could be heard. In rare cases they may be recognised as early as the eighteenth week. They are generally compared to the sounds of a watch ticking under a pillow. The average frequency is from 135 to 140 per minute. They are less frequent in large than in small children. The intensity of the sounds increases with the development of the child, but there is little change in their rapidity from the time they are first noticed till the end of pregnancy. The point of maximum intensity on the abdominal wall varies, of course, with the presentation and position of the fœtus. Frankenhäuser's statement that the average rate of pulsation is greater in the female than the male is now considered to be erroneous.

Uterine Souffle.—A second sound heard from the fifth month onward is of the nature of a soft blowing murmur synchronous with the maternal pulse. It is due to the passage of the blood through the dilated and tortuous uterine arteries, and is therefore best heard over the sides of the uterus. The intensity varies very much in different cases, but it is to be heard almost constantly after the fifth month. As a similar bruit is found very often in fibrous and occasionally in ovarian tumours, the uterine bruit is not alone of great value in diagnosing pregnancy.

Funic Souffle.—A soft bruit synchronous with the *fœtal* heart may also occasionally be heard. It is supposed to be due to some disturbance of the circulation in the cord produced by pressure.

The Changes in the Vagina in Pregnancy. —During early pregnancy, in the great majority of cases, the mucous membrane of the vulva and vagina is of a pale-violet colour, changing to deeper bluish tinge in the later months. It is noticed often as early as the second month, and is generally known as Jacquemier's test, and was first pointed out in 1837. The cause is a stasis of blood in the dilated vaginal blood-vessels. It is not a constant sign. Chadwick found it absent in 17 per cent of cases at the end of the third month. It is, however, usually present, and is a valuable corroborative sign in the early months when diagnosis is most difficult. Nor is it an infallible sign, for a similar discoloration is sometimes got in cases of fibroid and ovarian tumours, especially when these are impacted in the pelvis, and are obstructing the venous circulation in the pelvis.

Softening and Enlargement of the Vaginal Portion of the Cervix.—To the expert in vaginal examination these changes have a high diagnostic value from the third month onward. It has been stated that the absence of these changes in the cervix in a doubtful case of abdominal swelling reaching beyond the umbilicus may be taken as almost certain evidence of the non-existence of an intra-uterine pregnancy. The softening of the cervix, which is got in the early stages of the cervical catarrh, and sometimes in intramural fibroid tumours, is not likely to be mistaken for the softening that occurs in pregnancy, except in the early months when it is not well marked.

The Softening and Compressibility of the Lower Uterine Segment.—This was first drawn attention to by Hegar in 1884, and is generally known as Hegar's sign. It can be noticed best if the lower part of the body of the uterus is grasped between two fingers—one in the rectum, the other in the vagina—when the difference between the comparatively hard cervix and upper part of the uterus on the one hand, and the soft lower uterine segment on the other, is felt to be very marked, and the compressibility of the lower uterine segment is easily made out. The softening and broadening out of the lower uterine segment can be made out also by simple vaginal examination through the anterior and posterior fornices. To the expert Hegar's sign is of great value in the diagnosis of early pregnancy.

Changes in the Uterus.—The changes in the

size, form, position, and consistence of the uterus while it is still within the pelvis are the most reliable signs we possess for the diagnosis of early pregnancy.

Given favourable conditions for making a bimanual examination of the uterus in early pregnancy, *i.e.* a lax abdominal wall and uterus in its normal position, the doughy softness, the fluctuation, the alternate relaxations and contractions of the pregnant uterus can hardly be mistaken for anything else.

Still, mistakes are made, and made frequently; therefore, while the uterine tumour is still within the pelvis, it is well not to be definite in an *expressed* opinion, at any rate, after a single examination.

The Differential Diagnosis of Pregnancy

The increase in the size of the uterus being one of the most reliable signs in the diagnosis of early pregnancy, the conditions which are apt to be mistaken for it are naturally those which are also associated with this same sign. They are as follows:—

(*a*) *Chronic Metritis: Sub-involution.*—The uterus in metritis, though enlarged, will be felt to be uniformly hard, quite distinct from the doughy consistency of an early pregnancy. It is generally also associated with some degree of tenderness. Menstruation is profuse, and a history of some previous pelvic troubles will generally be obtainable.

(*b*) *A small interstitial fibroid* may be differentiated from pregnancy in the same way. It is much more difficult to differentiate between a small fibroid and chronic metritis than to distinguish between these conditions and pregnancy.

(*c*) *Enlargement of the uterus from retained menstrual or watery fluid* presents often much greater difficulties. *Retention of menstrual fluid* occurs in the great majority of cases in young girls in whom there is a congenital atresia of the cervix, vagina, or hymen. The uterine tumour which is found on abdominal palpation closely simulates, because of its doughy consistence, the pregnant uterus; but the fact that the patient has never menstruated, the pain which is generally present, and the absence of mammary signs, will generally excite suspicion and lead to a vaginal examination, when, if the atresia is in the hymen or vagina, the real nature of the case will be at once recognised.

Hæmatometron may, however, be got in women who have menstruated. The cause in them will generally be found to be an atresia of the cervix. This atresia may be inflammatory in its origin, or be due to the application of escharotics to the cervix. A history of pain each month, the absence of mammary signs and ballottement, and a want of correspondence between the size of the tumour and the period of amenorrhœa as in the former case, will generally excite suspicion, though the use of a speculum or an anæsthetic may be necessary to enable us to make a positive diagnosis.

Distension of the uterus by a watery or mucous fluid is a rare condition. It is met with after the menopause, and is due to senile changes, producing an atresia of the cervix. Occurring as it does in old women it is not likely to be mistaken for a pregnancy.

In the differential diagnosis of these conditions, which may simulate pregnancy in the early months, it will be seen that we are largely dependent on the bimanual examination of the uterus. The necessity for caution in our expressed opinion at this period has already been stated. When from any reason the uterus cannot be satisfactorily palpated bimanually—it may be owing to the thickness or tension of the abdominal wall, or to a backward displacement of the uterus—there is still greater reason for being careful. Pregnancy in a retroverted uterus in the early months is very easily overlooked even by the most expert examiner.

From the fifth month onward pregnancy has to be differentiated from all those conditions which are accompanied by enlargement of the abdomen. The most important of these are—

Ovarian Tumour.—Cystic ovarian tumours, from their comparatively rapid growth, from their consistence, and from the fact that not infrequently they are associated with amenorrhœa, have frequently been mistaken for pregnancy. The converse is equally true. The following points should be remembered in differentiating the one from the other. Ovarian tumours are, as a rule, slower in growth than the pregnant uterus, and while in the latter the abdominal swelling is central from the first, in the case of the latter we can generally elicit a history that the swelling was first noticed at one or other side. If amenorrhœa is present with an ovarian tumour, it is very unlikely that the size of the abdominal swelling will have the same relation to the period of amenorrhœa as that of the pregnant uterus. Menstruation, however, is regular in the majority of cases of ovarian tumour found before the menopause. Secretion may be present in the breasts in cases of ovarian tumour, but only rarely is this so, and then the other mammary changes are absent. Examination in the case of ovarian tumours for the uterine bruit, fœtal heart, and ballottement, will give negative results, and the uterus on vaginal examination can generally be felt not enlarged and displaced backward, forward, or to the side.

It is in cases of *hydramnios*, with consequent great abdominal enlargement, that mistakes are most apt to occur. Here several of the abdominal signs of pregnancy already mentioned may be absent, *e.g.* the fœtal heart and uterine bruit, and the active and passive movements of the fœtus. Vaginal ballottement, on the other hand, can be made out unusually distinctly, and

is the most reliable sign we possess for the diagnosis of this condition. It may be noticed in passing that the sound may be passed into the pregnant uterus without rupturiug the membrane, so that this simple operation must not be relied on to differentiate a pregnancy from an ovarian tumour.

Fibroid Tumours of the Uterus.—These, as a rule, are denser and grow more slowly than the pregnant uterus, and menorrhagia is generally a prominent symptom. In rare cases there may be found present mammary changes, but these are never so marked as those of pregnancy. A uterine bruit is heard in more than half the number of cases, but the fœtal heart sounds are of course absent, as are also the vaginal changes found in pregnancy. These points will generally serve to differentiate the one condition from the other. It must be kept in mind, however, that soft fibroids, and those undergoing cystic degeneration, may undergo a rapid increase in size, and that in soft fibroids the intermittent softening and hardening of the uterine wall so markedly felt in pregnancy may in rare cases also be noticed. In the absence of a very definite and reliable history it is very difficult to distinguish between a pregnancy in which the fœtus has died and been retained *in utero*, and a fibroid tumour.

Hydatid degeneration of the chorion, producing an irregularly shaped uterus and giving rise to irregular hæmorrhages, may closely simulate a soft fibroid tumour. The history, however, will be different in the two cases, and if this is not obtainable the lapse of a month will usually clear up the diagnosis.

Where a fibroid or an ovarian tumour and pregnancy co-exist the difficulty in diagnosis may be very great, but if the possibility of this occurrence is kept in mind, it will help occasionally in the diagnosis of an abdominal swelling with an obscure history and apparently contradictory symptoms.

Distended Bladder.—An over-distended bladder is not likely in itself to be mistaken for the pregnant uterus, though where it is present along with a pregnancy it may lead to certain errors in diagnosis. A distended bladder, for instance, the result of an impacted retroflexed gravid uterus, may be mistaken for hydramnios, or may cause one to suppose that the gestation is much further advanced than it really is. Such a mistake could only arise when a vaginal examination had not been made.

Obesity.—Accumulation of fat in the abdominal wall or in the omentum may not only render the diagnosis of pregnancy difficult, but may be mistaken for it. In women who are getting fat, menstruation frequently becomes scanty and sometimes ceases altogether. This obesity is very often developed at the menopause. The increase in size of the abdomen and the cessation of menstruation naturally suggest pregnancy, but when other corroborative signs are sought for in the mammæ or vagina they will be found absent. Still, in stout women caution should be observed in expressing a negative opinion, for obesity obscures most of the abdominal signs of pregnancy, and also renders the vaginal examination very difficult.

Spurious Pregnancy.—This is a rare condition, met with usually in neurotic patients, or in women who have married late in life and are desirous of becoming pregnant. In it all the symptoms and several of the signs of a true pregnancy are present. Menstruation may stop, the breasts secrete milk and enlarge, and there is morning sickness. Later there is swelling of the abdomen, quickening is felt, and the patient passes into a spurious labour. If suspicion is aroused, and a careful examination made, a mistake in diagnosis is not likely to be made. A resonant percussion note over the abdominal swelling alone excludes a true pregnancy. Should the patient be stout an anæsthetic may be necessary. In narcosis the abdominal tumour generally subsides.

Tumours of other Abdominal Viscera: Liver, Spleen, Kidneys.—These are not so likely to be mistaken for pregnancy as tumours of the ovary or uterus. Their growth is usually slow, and, with the exception of the abdominal swelling which they produce, they are unaccompanied by any of the other signs of pregnancy. The lower border of such tumours is free, and the edge of the hand can be passed between it and the pubis, thus proving their abdominal and not pelvic origin.

Ascites.—Ascites could hardly be mistaken for pregnancy. Fluctuation is very distinct, the character of the percussion note changes with the position of the patient, and the signs of pregnancy are absent.

The Diagnosis of the Probable Date of Confinement.—The date of delivery can be predicted only approximately, firstly, because the duration of human gestation varies within certain limits, and, secondly, because even if this duration was constant, we are unable to determine exactly when fertilisation of the ovum takes place. We do know now that ovulation occurs irregularly, and not only in relation to menstruation as was formerly supposed, and from observations in the lower animals it is probable that an interval of from one to fourteen days may elapse between insemination and fertilisation. So far as we can determine, the average duration of human pregnancy is from 270 to 280 days. It is found also that the first week immediately following menstruation is the most favourable time for fertilisation, and this fact is made use of in calculating the date of the onset of labour. Naegle's method, because of its simplicity, is the one generally used. It consists in going back three months from the date of the

beginning of the last menstruation and adding seven days; *e.g.* the date of the commencement of the last menstruation being August 7, labour may be expected about the 14th of the following May. Where the menstrual history of the patient is available no more accurate method can be got; but an error of from one to three weeks even with this is possible. The possibility of menstruation in the early months of pregnancy must always be borne in mind. Very often, however, as in cases where menstruation is irregular or absent, as in lactation, anæmia, phthisis, before puberty, etc., this method of calculation is impossible.

The Date of Fruitful Coitus.—This datum is rarely available in married women, though some profess to know from their sensations during coitus if conception has taken place. It is useful, however, in those cases where there is a history of a single coitus. The onset of labour may then be predicted at the 275th day after coitus.

The Date of Quickening.—Quickening generally occurs about the third term of pregnancy, and may therefore be used, in the absence of more reliable data, in calculating the probable date of confinement. In an intelligent multiparous woman it is of distinct value for this purpose. In all cases it is useful as a check where the menstrual history is indefinite, and is probably the most reliable datum available where from any cause menstruation has been suspended before impregnation.

The Height of the Fundus Uteri.—This depends to a considerable extent on the amount of liquor amnii and the size of the fœtus. Roughly speaking, however, it may be said that the fundus can be felt just above the symphysis pubis in the fourth month, at the fifth month midway between the symphysis and umbilicus, at the sixth month at the level of the umbilicus, at the seventh month two finger-breadths above the umbilicus, at the eighth midway between the umbilicus and the ensiform cartilage, at the ninth it reaches the ensiform, and in the tenth month the fundus sinks somewhat to a lower level. The level of the fundus, in many cases, does not vary much in the last weeks. The condition of the cervix at this time, however, will enable us to tell at least if labour is imminent, as it undergoes a *real* shortening, and its canal becomes merged into that of the uterus in the last fortnight of pregnancy.

The Length of the Fœtus in Utero.—This, according to Carl Braun, forms one of the best data from which to estimate the time of pregnancy. As it lies folded up in the uterus, the length of the fœtal axis is found to be about one inch for every lunar month of gestation. The measurement is got by applying one arm of a pair of callipers to the lowest part of the head, the other to the highest part of the breech. In primiparæ, where the head is already within the brim, it is necessary to introduce the lower blade into the vagina. A measurement of seven inches got in this way would indicate a seven months' gestation. This method of calculation is not, however, likely to be much used in general practice.

In calculating the onset of labour we will then generally rely on Naegle's method. It is well never to be too definite, a considerable margin should always be allowed, as mistakes of from one to three weeks are common.

Diagnosis of the Death of the Fœtus.—The indications of intrauterine death may be briefly stated as follows:—

1. The disappearance of the fœtal heart sounds if these have been previously noticed.
2. The cessation of active fœtal movements.
3. A slow diminution in the size of the abdominal tumour.
4. Diminution in size and tension of the mammæ.
5. Symptoms on the part of the mother of malaise. Chilliness and a feeling of dead weight in the lower abdomen.
6. Fœtid discharge from the uterus if the membranes have been ruptured.
7. Looseness and mobility of the fœtal cranial bones and feet on vaginal examination if the finger can be introduced within the internal os.

If the membranes remain unruptured the liquor amnii is slowly absorbed, and the dead fœtus may be retained *in utero* for weeks and even months. Such a consideration, as already stated, in the absence of a definite and reliable history, may present great difficulty in diagnosis.

The Diagnosis of Primiparity and Multiparity.—Under certain circumstances, generally medico-legal, it may be necessary to determine if a woman is pregnant for the first time or has previously been so. Traces of a former pregnancy which has gone to full time or past the sixth month can generally be found in the lax and pendulous condition of the mammæ, the relaxed abdominal wall, the lineæ albicantes, the absence of the hymen, the patulous condition of the vagina and vaginal orifice, and the lacerated cervix. A pregnancy, however, which has terminated in an abortion before the sixth month may leave no traces at all by which it can be subsequently recognised. It must also always be kept in mind that the lapse of a few years may obliterate many of the above signs, and that certain pathological conditions of the cervix in a primipara may closely simulate the characters of the cervix in a multiparous woman.

Diagnosis of Multiple Pregnancy. (*See* Multiple Pregnancy.)

Multiple Pregnancy

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1. *Definition and Varieties.*—One child at a birth is the natural or normal law in woman, but plural conceptions are not infrequent. The pregnancy is then spoken of as multiple. Two at a birth is comparatively common as compared with higher numbers, and is taken as the type of plural conceptions. Cases of three are rare, once in about 7000 deliveries; four still more rare, and of five not more than six or seven cases have been recorded. More than five at a birth has not been reliably authenticated.

2. *Frequency.*—The proportion of twins to single births may be regarded generally as 1 in 80 deliveries. Variations in frequency are shown in the statistics of different countries, from 1 in 60 (Bohemia and Ireland) to 1 in 100 (France), or 1 in 120 (New York). These apparent differences, however, may, to a large extent, be due to different systems of registration, and to defects, such as omission of abortions, in the registers. The frequency is no indication of the comparative fertility of the respective nations.

3. *The influences which predispose to twin-bearing* have attracted considerable attention, but the conclusions arrived at reach only a varying degree of probability. There is abundant evidence to show in many cases a *hereditary* influence, and also to support the opinion that there is an association between *twin-bearing and prolificacy*, but these influences, in all probability, apply to one class of twins only, the binovular, and not to the monochorionic or homologous twins.

The influence of the mother's *age and the number of the pregnancy* have been carefully worked out by Dr. Matthews Duncan. The following statements indicate the results of his investigations regarding the points in question:—

(1) The mean age of twin-bearing mothers is greater than that of mothers generally.

(2) While of all births three-fifths occurred among women under thirty, there occurred only two-fifths of the twins among the younger women.

(3) From the earliest child-bearing period till the age of forty is reached, the tendency to twin-bearing increases—or, in other words, the proportion of mothers of twins to mothers of the same age increases as the fertile women grow older.

(4) The same is found as regards the initial fertility. Newly married women are more likely to have twins the older they are. Every one hundred and fifty-third woman among the youngest fertile women bears twins within two years after marriage; among the older women, from thirty-five to forty years of age, every forty-second woman bears twins within two years after marriage, or nearly four times as many.

(5) As regards the number of the mother's pregnancy, the proportional frequency of twins increases with the number of the pregnancy. To this general statement there is an exception in the case of first pregnancies. Woman has apparently an increased chance of bearing twins in her first pregnancy.

Influence of the Male. — Cases have been recorded which appear to support the view that the male may exert some influence in the procreation of twins; as where a man twice married has had twins by both his wives; or where a marked hereditary tendency to twin-bearing has been traced in successive generations, it has manifested itself in the family of one of the males. Seeing, however, that twins must primarily depend on ovulation, such instances may be regarded as mere coincidents, unless it can be shown that the generative vigour of the husband can stimulate the function of ovulation in the wife.

4. *Origin and Development.*—Twins may be developed from two ova, *binovular*, derived from either a single Graafian follicle, or two separate ones, in the same or different ovaries; or they may take origin from one ovum, *uniovular*, containing a double germ.

Ahlfeld has advanced the view that twins may take origin from division in the formative material within a single area germinativa. When the division is complete, separate twins are produced with a single amnion, or if incomplete, conjoined twins or some form of double monster is the result.

Evidence of the origin and mode of development can be obtained by making a careful examination of the placentæ and membranes. This should always be done, especially where anything peculiar in the fœtuses or labour has been observed. The want of any note as to the relation of the membranes to the fœtuses has much impaired the value of the record of cases. In certain conditions the relationship may be difficult to determine. The separability of the chorion from the amnion at full term and the difference in transparency make easy the recognition of the envelopes. During examination the structures should be placed under water.

When *binovular* there are *two chorions*, *dichorionic*, and *two amniotic sacs*, and if the ova have been embedded in the mucosa sufficiently apart, each will have had a separate decidua reflexa, but when close together the latter may have been in common.

When *uniovular* the chorion is single, *monochorionic*, but with two amniotic sacs in nearly every case. Occasionally both fœtuses lie in a common amnion. A single chorion occurs once in eight cases, a single amnion once in 132 twin pregnancies (Ahlfeld).

5. *Examination of Placentæ and Membranes.*—*a.* When the placentæ are separate, and each fœtus lies in its own chorion and amnion, there can be no doubt of their origin from two distinct ova. Although developed separately the placentæ, in the process of growth, may come in contact and so combine as to give the appearance of a common placenta. The double chorion will indicate the distinct origin. In such cases the placental vessels are entirely distinct, there is no anastomosis.

b. If only one chorionic covering is present the fœtuses have been derived from a single ovum with two germs. In such cases the placentæ are always united, and it is not rare for anastomosis to have existed between the two circulations. The cords may be inserted close together or widely apart.

c. In such cases, with a single chorion, there is nearly always two amnions. Both fœtuses, however, may be found to have occupied a common amnion. The sac may have been single from the first, for the blastoderm may envelop both fœtuses in a common amnion; but in other cases it would appear, that though primarily double, the cavities have been reduced to one by the absorption of the septum or portion of the two sacs in contact. Remnants of the septum have been found, and traces may be detected, between the insertions of the cords, in the forms of lines of fat from degeneration of the tissues and adhesion of the amnion beneath.

Homologous Twins. — This term has been applied by Ahlfeld where normally formed twins of the same sex have been enclosed within one chorion and drawn their nourishment from a single placenta—*the monochorionic disomata* of teratologists. They are strikingly alike, and present great similarity in bodily development and even in disease.

A very different result is found in another group of this class, the *allantoido-angiopagus* (Ballantyne) or *omphalo-angiopagus* of other writers. They are defined as "twins of the same sex, enclosed within a single chorion, one of which shows more or less grave developmental defects, and communicates with the other by means of the vessels of the umbilical cord in the neighbourhood." The modes by which this anastomosis occurs are very numerous, they are both within and without the placenta, and it thus becomes possible that the circulation of the deformed twin, which may not have a heart, is maintained by the heart of the normal twin. Hence the importance of carefully describing the vascular arrangement in all cases of twin monstrosities, and of including the placenta and membranes when sending a specimen of deformity to a specialist.

6. *Sex of Twins.*—Monochorionic twins are always of the same sex. When the chorion is double they may be of the same or different sex. Males preponderate over females, but to a lesser extent than in births generally. Most frequently the two children are of different sex. In the Prussian statistics, in 150,000 twin pregnancies, a boy and girl occurred 54,000 times, both males 50,000, and both girls 46,000 times.

7. *The Diagnosis.*—The presence of more than one fœtus *in utero* may at times be suspected from the size and form of the uterine tumour, but a certain diagnosis can be made before labour only on the evidence of palpation and auscultation. The uterus need not necessarily be large if the twins are small, or there is great disparity in size; and a large uterus may be due to an extra-sized single fœtus, or to an excess of liquor amnii. The evidence of a large quantity of the waters, on the other hand, does not negative twins, for that condition is not infrequently present in plural pregnancies. In the earlier months there is no certain sign. When pregnancy is sufficiently advanced to permit of the fœtal parts being recognised by palpation, the definite character of a *head* may be made out *in two places*, but the apparently numerous points of *fœtal limbs* are liable by themselves to deceive. The *fœtal heart sounds* heard readily in two places are insufficient; but when two points of greatest intensity are found at some distance and on different sides of the uterus, with a line of diminishing intensity and again increasing between, or the sounds lost and again found, the inference of plural pregnancy is very strong, and is made certain if on two or three observations made simultaneously by two observers, the rate of the heart-beats proves to be different at the two spots.

8. *The course of pregnancy* is more liable to be disturbed when plural than when single. Abortions are comparatively more frequent, and there is an increased tendency for labour to ensue more or less prematurely. This does not arise, as is generally supposed, from the over-distension and greater irritability of the uterus, but from the feebler organisation and consequent earlier occurrence of the physiological degenerative changes which are now known to precede the onset of labour.

When the fœtuses are developed from two distinct ova, *dichorionic*, the prospects are much more favourable. Inequality in development and growth is common—one fœtus, as a rule, is more favoured than the other. The relative position in the uterus is an insufficient explanation, for the most favoured is not always the first to be born. In the *monochorionic* variety defective development and nutrition are more likely to occur, and to lead to malformations or death of one of the fœtuses. The degree of proximity of one embryo to the other, their relation to the decidua serotina, and the union of the two allantoides, are the chief factors in causing interference. The greatest diversity is found where both fœtuses derive their nourishment from one common placenta.

One fœtus may die and be retained without undergoing maceration or decomposition. It is expelled and found amongst the membranes, flattened and shrivelled, the *fœtus papyraceus v. compressus.*

Well-authenticated cases have been recorded where one fœtus has aborted and been expelled, the pregnancy, nevertheless, continuing with the other and reaching maturity at the full time.

In the later months a considerable interval of days and even weeks in a few cases has elapsed between the births of the two children. Some of these have been proved to be due to *uterus duplex*, and probably all are. Such an occurrence, and the occasional great disparity between the two children, gave origin to the hypothesis of *superfœtation*, by which is meant the possibility of a woman being capable of conceiving a second time when already pregnant for one or more months. Excluding ectopic gestation and the presense of a double uterus, such a hypothesis is now held to be "physiologically untenable." *Superfecundation*, or the impregnation of a woman a second time, by two separate sexual acts within one intermenstrual period, is totally different, and has been proved to occur by cases where the fathers of the twins have been of different races. But the short interval within which such second impregnation must take place does not explain the great disparity in development, nor the greater interval that has occurred in some cases between the separate births.

Management of Pregnancy

Pregnancy, besides producing local changes in the sexual apparatus, is the cause of a widespread alteration throughout the whole of the body. The organs of a pregnant woman have to adapt themselves to a changed order of things (*see* "Physiology of Pregnancy"). Possibly the most important change is the conspicuous alteration in the quality of the blood and the increase in its quantity, this throwing an increased strain on all the excretory functions—lungs, kidneys, bowels, skin. If any of these organs are not equal to the increased pressure put upon them, symptoms, moderate or severe, of toxæmia supervene (p. 227).

The main indication for management thus lies along the lines of maintaining as good a hygienic state as possible, and when any organ is known not to be quite sound, to assist it as far as possible to stand the extra strain.

When a pregnant woman is healthy and leads at ordinary times a sensible hygienic life, nothing is better than that she should adhere to the same mode of life as suited her well before.

Regular exercise in the open air is most important, and should be insisted upon. It is undoubtedly injurious to lie in bed most of the forenoon, and rest on a sofa in the afternoon. Such practices are only justifiable in a healthy woman, at the date at which a menstruate period is due, if there is a history of previous abortions. This "over taking care" brings on indigestion and sleeplessness, and also loss of tone in the muscles, which makes the labour more prolonged, and brings about a loss of recuperative power during the puerperium. We have a good object-lesson in the wives of working-men, who continue to work until labour commences, without detriment to themselves.

Violent exercise of any sort tends to produce miscarriage. Driving over rough roads, prolonged railway journeys, dancing, and bicycling, except in the early months, should be forbidden. The lifting and carrying of heavy weights must be avoided; while the continuous use of a sewing-machine or systematic massage are also not advisable.

The increased elimination of carbonic acid gas makes it imperative that the patient should have plenty of fresh air; thus crowded churches theatres, concerts, balls, and crowded, stuffy rooms should be avoided. Going to bed early and sleeping in a large, well-ventilated room are important. Mental excitement should be avoided. Irritability of temper requires great forbearance on the part of the patient's friends.

Clothing.—The enlargement of the breasts and abdomen and the increase of the cutaneous vascular system make it necessary to have a change in the clothing. Light warm garments to prevent chilling of the body, and roomy clothes that do not interfere with the respiration and circulation, are required. The dress should be loose and easy, with no tight waistband. After the three and a half months the ordinary corset should be discarded and special corsets used, which support the enlarging breasts without pressure on the nipple, and which also have a band at the lower edge which can be let out on either side with laces as the abdomen increases in size. In multiparæ with flaccid abdominal walls, a belt will be found of great comfort; it can be made of flannel or lint, and made to fit.

Tight garters are injurious, stocking suspenders being used instead; the sooner these are used the better chance of avoiding troublesome varicose veins. After the fifth month, when the increasing abdomen lifts the petticoats off the lower part of the abdomen and thighs, loose flannel drawers should be worn to prevent chills.

Diet.—As far as our knowledge at present takes us, the diet of a pregnant woman should be a good general mixed diet of plain, well-cooked, simple food, taken at regular intervals. The quantity of nourishment need not exceed what is usually required by a healthy woman, but it is well to take food in smaller quantities and more frequently during the later months, when the enlarged uterus presses against the

stomach. "Longings," so far as they are kept within reasonable desires, can, of course, be gratified; but no evidence is obtainable that the refusal to satisfy unreasonable caprices has any influence mentally or physically on the child. Strong alcoholic and malted drinks are unadvisable, but there is no reason to prohibit a little light wine at dinner and lunch, if such has been the habit of the patient previously.

That the diet must directly influence the growth and development of the fœtus *in utero* is reasonably clear. Recent scientific experiments seem to show that possibly at no very distant date we may learn in what manner the growth and development of the bones of the fœtus and the tissues of the mother may be favourably modified by a special dietary, so that labour may be shortened and made more easy for the mother, and less dangerous to the child. Indeed there are indications that diet may so influence the maternal and fœtal tissues, so as to lead to the birth of a viable child in cases where the previous obstetric history indicated the improbability of this ever taking place.

Two suggestive cases have come under the writer's notice. The first was a multipara who had had several confinements, some of them premature, but never a living child, owing to the size of the pelvis. When I saw her she was on a vegetarian diet. The labour came on prematurely at the eighth month, and she was easily delivered of a healthy infant which lived. The second case was a primigravida, æt. about twenty-six, a very small woman, who was a rigid vegetarian (from conviction); she was in the second stage of labour before feeling anything, and the infant was born very rapidly, certainly an exceedingly easy labour for a primipara of her age. It is, of course, possible, but I think unlikely, that these were merely coincidences.

It is probable that the diet which will ultimately be found to exert a beneficial influence in this direction will be one in which the carbohydrates and fats enter most largely. If space allowed, many facts could be cited from comparative obstetrics, and also from the obstetric histories of different races, in favour of this view. With our present knowledge it is sufficient to suggest that this line of investigation should be studied carefully from the clinical side no less than from the standpoint of experimental laboratory observation.

The Regular Evacuation of the Bowels is very important; during pregnancy there is always a tendency to more or less constipation. This difficulty is best got over by giving a liberal supply of fruit. If medicine is necessary, some of the mild vegetable purgatives are to be recommended, cascara sagrada, liquorice powder, or tamar indica lozenges. The constant use of mineral waters tends to aggravate hæmorrhoids, which are often very troublesome, and strong cathartics are not safe as they may be the cause of a miscarriage. Castor-oil is a safe but to most people a very disagreeable remedy. Attention to this point is probably the most important factor in the prevention of many of the minor and even major manifestations of toxæmia in pregnancy.

During the early months complaints of inability to pass water, or constant desire to micturate, should put the practitioner on his guard for cases of retroversion. The urine should be examined from time to time, especially in the later months, and more particularly in primiparæ. If albumen be detected, special precautions must be adopted (*see* "Pregnancy, Pathology of").

The *skin* requires special care. It should be kept in good condition by frequent bathing. When acting properly it is able to relieve the kidneys of a portion of the extra strain thrown on them. Women who are in the habit of taking a daily cold bath should continue to do so, and should take in addition a warm bath once a week (90°-96° F.); those who are not accustomed to the daily bath should have a tepid bath twice a week (77°-90° F.). During the last six weeks the warm baths may be taken three or four times a week; this is specially good for primigravidæ.

The increased vaginal secretion renders it important for the external genitals to be frequently cleansed. These can be washed morning and night with tepid water and a good lather of soap, a little spirit and water rubbed in afterwards; an advantage, as it prevents any heavy odour. If the vaginal discharge becomes irritating, either causing pruritus or excoriations, the vaginal douche may be used. The quantity injected should not exceed a pint of water, should be injected very slowly, and only lukewarm water used. Chafing under the breasts and groin can be got rid of with washing twice daily with vinegar and water, drying carefully, and dusting on boracic acid.

The *breasts* should be kept warm and supported without any undue pressure upon them. The nipples and areola during the last two months should be hardened by regular washing with water and alcohol. The methods of bringing forward depressed nipples, such as pressing back the areola with elastic rings or a nipple shield, or pulling forward the nipple, usually give a good deal of pain, and it is doubtful if they do any good.

Sexual excess in young women is often the cause of early abortions, and abstinence is advisable.

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Affections of the Ovum and Decidua

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Cysts

The nomenclature of the subject is so varied that a short restatement of the structure of the ovum and decidua during early pregnancy is needed in order to avoid repeated definitions in the course of the following discussion.

The lining membrane of the uterine cavity consists of a delicate stroma containing round connective tissue cells. This tissue is placed directly upon the uterine muscle, is richly supplied with small blood-vessels, and is perforated by numerous pits or crypts. Its free surface is covered by a single layer of columnar epithelial cells, which dips down into and lines completely the crypts, follicles, or glands of the uterine mucosa. It is this membrane which, during pregnancy, undergoes certain definite changes and forms the decidua. While the mucosa is about $\frac{1}{20}$ in. thick just after menstruation, it grows until it is about $\frac{1}{6}$ in. thick before each menstrual flow begins. When pregnancy begins and arrests menstruation, the lining of the uterus continues to grow until it reaches its maximum thickness of about $\frac{1}{3}$ in. during the third month of gestation. The so-called glands dilate, and the blood-vessels also enlarge and multiply. The round connective tissue cells proliferate rapidly, and many of them increase greatly in size and assume irregular shapes, thus becoming the "decidual cells" characteristic of the mucosa of pregnancy. These decidual cells are most numerous near the free surface of the membrane, which soon loses its covering of epithelial cells. The epithelium lining the glands also degenerates, and for the most part disappears, except from their deepest portions, where it remains alive in sufficient quantity to secure the regeneration of the epithelium of the whole uterine cavity after the end of pregnancy. Three layers can be recognised in the uterine mucosa as modified by pregnancy: (1) A superficial compact layer, with many decidual cells lying between the glands, which are not much dilated, but whose epithelium is flattened, degenerated, and largely lost; (2) A spongy or reticulated layer, whose glands are very widely dilated, and their epithelium much degenerated, some decidual cells, and many spindle cells and fibrils of connective tissue occupy the spaces lying between the dilated glands; (3) A deep layer of delicate connective tissue containing the undilated blind ends of the glands, whose epithelium is unchanged. On examining a pregnant uterus at any time before the end of the third month we find that a uterine cavity still exists, and is lined completely by decidual mucosa. The ovum lies embedded in the compact layer of the decidua on one wall of the cavity, its whole surface being in contact with the decidual connective tissue, and not with maternal epithelium. The decidua underlying the ovum—*i.e.* between it and the uterine wall—is called the decidua serotina, and is the site of the developing placenta. That covering the ovum, and lying between it and the uterine cavity, is the decidua reflexa. The rest of the decidua lining the uterine cavity, and not at first touching the ovum, is the decidua vera. During the third month the ovum, covered by the decidua reflexa, begins to occupy the whole space within the uterus. The reflexa and vera now come into close contact, obliterating the uterine cavity. The two become adherent, and blend into a single layer of tissue. After the third month the decidua becomes less vascular and pale in colour; it is flattened out and thinned by the pressure of the growing ovum. Fatty and other degenerations occur in the decidual cells. The vessels become thrombosed by the proliferation of their endothelium, and before full time is reached the decidua is reduced to a thin and unimportant layer of maternal tissue covering the ovum, and coming away with the secundines at parturition by separation through its superficial layer, leaving the deep layer with the embedded ends of the uterine glands as the sole covering of the muscular wall of the uterus. The ovum and decidua can most readily be studied by examining "complete abortions" expelled about the end of the second month of pregnancy. Such specimens consist of the ovum enclosed in the decidua. On removing the decidual covering (decidua vera and decidua reflexa, with the remains of the uterine cavity between them being recognisable as a rule) the ovum itself is seen to be a sac covered all over

with shaggy branching processes, the villi of the chorion. On opening the sac its cavity is seen to contain a little liquor amnii and the fœtus, while it is lined by the thin smooth amnion, which can easily be stripped from the thicker outer membrane, the chorion, which bears the villi on its outer surface. To understand the structure of these fœtal membranes, a portion of the wall of the ovum with the decidua attached should be sectioned for microscopic examination. The amnion is then seen to consist of two layers, the inner one being a single layer of epithelial cells (fœtal epiblast), while its outer stratum is of delicate connective tissue (fœtal mesoblast). The chorion is also composed of two layers, the inner of connective tissue, the outer of epithelium. The connective tissue layers of amnion and chorion are in contact with, and indeed continuous with, one another. (*See* Figs. 1 and 2.)

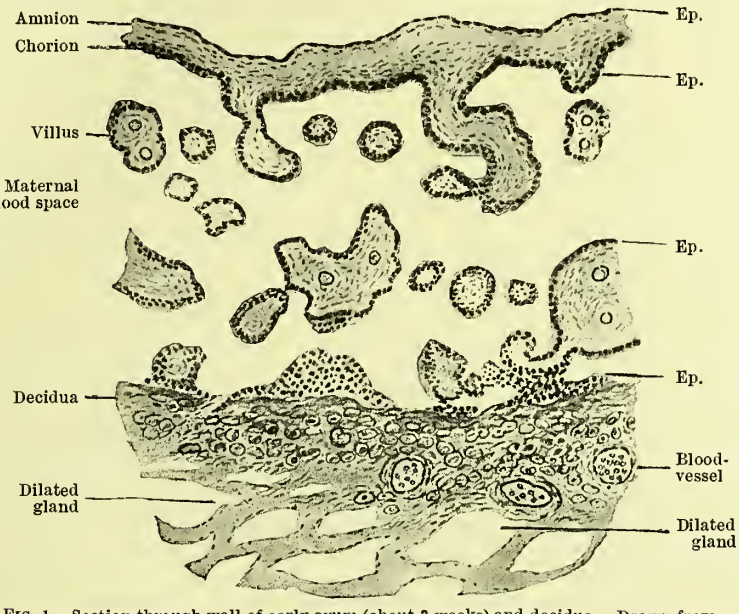


FIG. 1.—Section through wall of early ovum (about 3 weeks) and decidua. Drawn from nature. Zeiss ½ in. ob. oc. 3. Ep. = fœtal epithelium.

The fœtal epiblast, or chorionic epithelium, forming the outer stratum of the chorion, is a layer two cells thick. Each chorionic villus is an outgrowth of the chorion, being composed of a core of embryonic or mucous connective tissue covered by a layer of fœtal epithelium which is, like that of the chorionic membrane, two cells thick. The mesoblastic core of the villus is continuous with the connective tissue layer of the chorion, and contains capillary loops filled with fœtal blood corpuscles. The appearance of the surface of the decidua shows that the fœtal epithelium has the power of eating into and so destroying the maternal tissues. By this phagocytic action of their epithelium the tips of the villi penetrate and become attached to the compact layer of the decidua. By this means also the decidual blood-vessels are opened into and thrown into communication with the space between the chorion and the decidua which is crossed by the villi, so that the maternal blood circulates freely in the intervillous space. In many specimens it is possible to distinguish two cell layers in the fœtal epithelium covering the chorion and its villi. The outer layer shows no cell outlines, and it consists of a stratum of protoplasmic material containing numerous nuclei and vacuoles. This outer layer of the chorionic epithelium is known as the "syncytium." The inner layer, which is in contact with the connective tissue of the villi and chorionic membrane, consists of distinct cells of large size, and is known as the "layer of Langhans."

In order to understand the affections of the amnion, it is needful to remember that although the amniotic epithelium appears in transverse sections as a layer of apparently cubical cells, the appearance of the amnion when "silvered" and viewed in surface is closely suggestive of a serous membrane. The epithelial cells are flat and irregular in outline, while numerous stomata or openings between the cells are visible on careful examination.

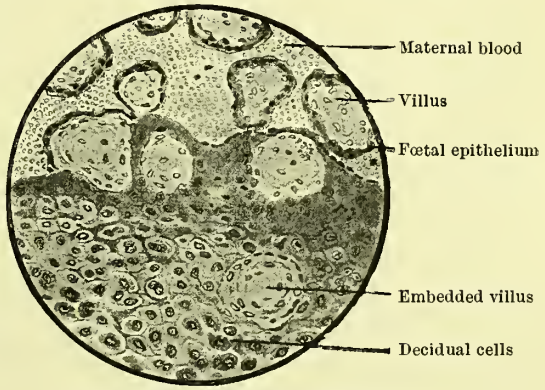


FIG. 2.—Decidua and villi at the third month. Drawn from nature. Zeiss ⅓ in. ob. oc. 2.

AFFECTIONS OF THE DECIDUA

Hypertrophy of the Decidua.—The changes that occur in the endometrium during early pregnancy are mainly hypertrophic in nature, and, like other physiological hypertrophic changes, they are liable to exceed normal

bounds and become pathological. Diffuse hypertrophy of the decidua consists in a general overgrowth of the maternal covering of the ovum. If slight, this condition simply leads to the production of a decidua which is thicker than usual at the end of pregnancy. If excessive, it may result in abortion at any period of gestation, but usually does so at the second, third, or fourth months. Microscopic examination reveals the presence of an excessive number of decidual cells, the connective tissue is unduly soft and yielding, and the vascular spaces are unduly large. Hæmorrhage occurs into the decidua or into the ovum itself on causes slighter than those necessary to produce it under normal conditions. Congestion due to backward pressure from cardiac lesions, coitus, the action of purgatives, and other slight accidents are liable to thus bring about the death of the ovum, more especially at times corresponding to the menstrual periods.

Polypoid Hypertrophy of the Decidua.—Virchow has described this condition under the name of *endometritis decidua polyposa vel tuberosa.* The whole decidua is thickened, but very unevenly, its surface being raised into polypoid excrescences, between which the openings of the uterine glands may be observed. The tissue is very vascular, and is rich in decidual cells. The condition is sometimes found, as in Virchow's case, in connection with syphilis, and generally with a persistent endometritis. Müller states that the chorionic villi frequently show myxomatous change. This condition affects early ova and ends in abortion, which occurs from the second to the fourth month.

Atrophy of the Decidua. — Spiegelberg, Priestley, and others have described atrophy of the decidua, causing the ovum to be attached by a pedicle instead of being embedded in a continuous layer of maternal mucosa. This condition, by lessening the support usually provided for the delicate membranes of the ovum, has resulted in rupture of its sac through the thinned decidua reflexa.

Inflammation of the Decidua.—Inflammatory changes in the decidua follow, as a rule, persistent endometritis. If acute, they tend to produce early abortion; and if chronic, they tend to cause fibrosis, undue firmness and toughness of the tissues which attach the ovum to the uterine wall. The result of this is difficulty in separation of the ovum from its bed. If expulsion of the ovum is premature, incomplete abortion occurs; if it is deferred till term, adherence of the placenta or membranes, or both, complicates the third stage of labour. Purulent inflammation of the decidua, due to microbic infection, has been described by Donat. A placenta expelled at term brought with it decidual masses infiltrated with pus, and purulent fluid was collected between the amnion and chorion, which were thickened and opaque.

The symptom known as hydrorrhœa gravidarum is due in some cases to a catarrhal inflammation of the glandular elements of the decidua following a chronic catarrhal endometritis affecting the epithelial linings of the uterine glands. Fluid containing mucus is secreted in excess, and accumulates between the decidua and the chorion. It may escape either in gushes or by a slow dribbling during the later months of pregnancy. This condition must not be confused with gradual escape of the liquor amnii, nor with cases in which this fluid collects between the amnion and the chorion, escaping suddenly on rupture of the latter membrane.

Leopold has demonstrated the occurrence of small cysts in the decidua of young ova, caused by the retention within the uterine glands of the secretion during early pregnancy. During the course of various infectious fevers, which frequently cause abortion, the decidua undergoes acute inflammatory and hæmorrhagic changes, which are held to be the means by which abortion is produced. Klotz has described these changes as they occur in measles, and Slavjanski records a condition of hæmorrhagic decidual endometritis in cholera. The decidua was thickened, dark purple in colour, and full of extravasated blood.

Syphilis has been found to exist in connection with almost every form of decidual disease; but there is nothing accurately known as to its effects on the decidua beyond the fact that it frequently causes abortion.

The above-mentioned affections of the decidua are seldom if ever diagnosed, and they admit of no treatment. While of pathological interest they have no clinical importance except in so far as they affect the nature and management of the abortions they cause. They are indirectly causal of many sequelæ of abortion and parturition.

Hæmorrhage.—Hæmorrhage into the decidua, however, sometimes causes the death of the ovum, followed by its prolonged retention in utero and its final expulsion in the form of a fleshy or bloody mole (*see also* "MOLE, MOLAR PREGNANCY," vol. vi. p. 438).

Fleshy or Carneous Mole.—When the ovum dies without the occurrence of hæmorrhage, and is then retained for a time in utero, it shrinks and undergoes fatty degeneration, being ultimately expelled as a shrivelled mass known as a "blighted ovum." When, however, bleeding occurs into the decidua, the intervillous space is distended, the decidua is more or less broken up. The subchorionic hæmatoma thickens the wall of the ovum and causes the chorio-amniotic membrane to be thrown into convolutions, which project as bosses and tuberosities into the amniotic cavity. Some specimens have been

described in which the amnion was separated from the chorion by blood-clot, the hæmatoma being subamniotic as well as subchorionic. The maternal blood occasionally breaks into and clots within the amniotic cavity itself.

The structure of the mole depends upon the age of the ovum at the time of its death, and upon the quantity and position of the blood-clot which is formed in and around it. Thus if the ovum dies before the formation of the placenta, and when the whole chorion is covered with villi, the whole wall of the ovum is thickened by the formation of blood-clot between the villi. If the placenta is already formed it becomes curled round on itself, so that its uterine surface forms the external surface of the mole, the membranes being crumpled up within its concavity. Again, if the hæmorrhage is sufficient to separate the decidua completely from the uterine wall, the structure of the mole differs from that found in cases where the decidua remains in contact with the muscular wall of the uterus.

Morgani is the earliest writer who shows a clear understanding of the morbid anatomy of retained apoplectic ova. Writing before the year 1859, Scanzoni states that the thickness of the chorion in these cases is due to the effused blood, more or less organised, which is found between the villi. This, he continues, becomes transformed into connective tissue, which becomes vascular and adheres to the uterine wall. Breus has made microscopic examinations in five cases. Berry Hart and the author have also described the histological changes observed. After the death of the ovum and the stoppage of the chorionic circulation the small fœtal vessels in the villi disappear, the larger ones being more slowly obliterated, and leaving for some time a few blood crystals and amorphous granules to mark their late position. The fœtal connective tissue between the epithelial layers of the amnion and the chorion, as also that forming the cores of the villi, is compressed, but is not otherwise altered for a long time. The fœtal epithelium lining the amniotic cavity also remains recognisable, but the outlines of its cells are lost, and the nuclei become clouded. The fœtal epithelium of the chorion and the chorionic villi undergoes similar changes. When this epithelial covering of the villi degenerates after the stoppage of the fœtal circulation, the maternal blood in the intervillous spaces does not long continue to move, but soon forms a firm blood-clot embedding the villi. This intervillous blood-clot consists so largely of fibrin that it seems probable that the degenerated epithelium of the villi acts as a foreign body, layer after layer of fibrin being deposited upon the villi from the maternal blood until the whole intervillous space is thrombosed. Eden has shown that this occurs in parts of the living placenta which lose their fœtal blood-supply towards the end of normal pregnancy.

As the liquor amnii is absorbed the subchorionic hæmatoma bulges inward into the cavity of the ovum, throwing the amnion and chorion into the characteristic bosses or elevations which are seen on cutting into the ovum, and laying open its amniotic surface. If recurrent hæmorrhages into the decidua separate the ovum completely from the uterine wall, the outer surface of the mass comes to be formed by concentric layers of blood-clot, and the tissues of the ovum may remain much as described above until the whole mass is expelled, it may be months after the death of the ovum.

In other cases the decidual hæmorrhage, while sufficient to kill the ovum and form the subchorionic hæmatoma, does not separate the ovum from the decidua by a thick layer of blood-clot, but leaves the mass of villi embedded in fibrin still in contact with the uterine wall. In such cases further changes are caused by the continued life and growth of the decidua. The decidual cells continue to multiply, and, exercising a phagocytic action, they attack the fibrin, the chorionic epithelium, and ultimately the connective tissue cores of the villi, making an attempt to convert the mass into tissue of the nature of decidua. This action of the decidual cells in organising masses of retained placenta has been described by Hartmann and Toupet, and they have applied the name *Deciduoma benignum* to a placental polypus thus converted into connective tissue. It is worthy of note that Cazeaux, Naegele, Osiander, and others believe that complete absorption of placental structures by the uterine mucosa has occasionally occurred. For the retention of dead ova in the uterus during long periods the absence of septic infection is a necessary condition.

Two factors are concerned, as a rule, in the production of these molar pregnancies. On the one hand is decidual disease, which renders extravasation of blood more liable to occur; and on the other hand there is usually some general condition which favours pelvic congestion. Such are cardiac lesions, traumatism, prolonged exertion, and the exanthemata. For the retention of the ovum it is essential that the uterine cavity remain free from septic infection.

Symptoms and Course.—The usual symptoms and signs of pregnancy appear and progress normally up to a certain point, they then pass away and leave the patient with amenorrhœa or with irregular vaginal discharges, sometimes resembling menstrual blood, but at other times consisting of mucus stained brown with blood pigments. The breasts cease to enlarge, and shrink to their original size. Pain, as a rule, is absent, though there may be down-bearing, backache, and general malaise. The uterus

becomes firmer in consistency than in normal pregnancy, the cervix losing its characteristic softening. The organ may remain the same size, may grow slowly, or may gradually lessen. For repeated hæmorrhages may increase its size by adding layer after layer of blood-clot to that already surrounding the retained ovum; or, in the absence of fresh hæmorrhage, the absorption of the liquor amnii may cause reduction in the bulk of the mass. Expulsion finally occurs spontaneously and accompanied by more or less pain and bleeding. Some ova have been retained in utero as long as eighteen months, but expulsion more often occurs within a much shorter time.

In the diagnosis of this condition everything depends upon the recognition of pregnancy, as, apart from this, the practitioner will be confused by thinking in turn of all the recognised causes of uterine enlargement. Given, however, a clear history of commencing pregnancy, followed either by a threatened abortion or by gradual disappearance of the signs and symptoms of pregnancy, there is still room for dubiety in many cases. It may be difficult, if the uterus continues to enlarge, to be sure that the fœtus is really dead, and Simpson points out the danger of interference in such cases, lest, while one of twins has perished, the other should be still alive. Vesicular degeneration of the chorion may also be suspected under these circumstances. If the uterus is decreasing in size it may be difficult to prove that any product of conception is still within it. Indeed, it is often impossible to ascertain the true condition of affairs until careful observation has been continued over a period of several weeks or even months.

The prognosis is good, as there is little disturbance of health as a rule, and the bleeding which occurs on expulsion is seldom severe. There is always the risk of sepsis, and there is always a marked tendency to subinvolution, the uterus remaining large and flabby much longer than after an ordinary abortion. In a few cases the condition known as deciduoma malignum has followed the expulsion of retained ova.

Owing to the difficulty in diagnosis, the management of the cases recorded has almost always been expectant. If, however, the condition is clearly recognised, the cervix should be partly dilated, and uterine contraction should then be secured by packing the vagina, or distending it with a rubber bag. The mole having been expelled naturally, or the uterus having been cleared out by hand, special precautions against subinvolution should be taken. Rest in bed, hot douching, and the exhibition of strychnine, iron, quinine, and hydrastis are of value in the after treatment.

The blood mole, or mola hæmatomatosa, is simply a variety in which blood-clot forms the bulk of the mass. The fibrinous mole of Berry Hart appears to be due to disease of the chorion, and is accordingly referred to under that heading.

Spurious Abortion.—This term has been applied by Eden to a condition of which he has described three examples. It is supposed that an ovum is fertilised, and develops far enough to set up the changes in the endometrium which usually accompany gestation. The ovum perishes at such an early date that it leaves no trace of its existence, but the decidual membrane goes on growing for two or three months, accompanied by amenorrhœa, enlargement of the uterus, enlargement of the breasts, and morning sickness. Brownish discharge or bleeding is then followed by the expulsion of a decidual cast of the uterus, similar to those expelled in cases of ectopic gestation, and containing no fœtal elements whatever.[1]

The cast is triangular in shape, and consists of a sac formed entirely of uterine mucosa, which has undergone the changes mentioned in describing the development of the decidua. Without careful investigation these casts would be mistaken for early abortions, and they are probably more common than is supposed. Whenever such a cast is expelled it is necessary to make sure that ectopic pregnancy does not exist, in order that the patient may be promptly treated for that condition if necessary.

This condition cannot be diagnosed clinically, and demands no treatment until expulsion of the cast occurs, when recovery is rapid and complete.

Malignant Disease of the Decidua.—No account of the affections of the decidua would be complete, in the present state of our knowledge, without reference to the form of malignant disease which is known (amongst other names) as deciduoma malignum. For although Veit and several British authorities hold that this condition is sarcoma of the uterus, existing before, but modified by the occurrence of pregnancy, a number of observers believe that the new growth begins in the decidua itself, is essentially a disease of pregnancy, and cannot be pre-existent to its occurrence.

This view was taken by Sänger, who first described a case in 1888. He considered it to be a sarcoma derived from the decidual cells during pregnancy, and later suggested the name sarcoma-deciduo-cellulare as more descriptive than deciduoma malignum.

The great majority of observers on the continent of Europe and in America, together with some British authors, hold that the growths in question are not sarcomata of maternal origin, but are fœtal carcinomata derived from the

[1] The author has, since the above was in print, described a case in which a uterine cast of this kind was expelled after eight weeks' amenorrhœa, together with a small body, the size of a split pea, which proved to be a complete ovum of about five days' growth. — *Trans. Obstet. Soc. London*, 1901.

chorionic epithelium. Having regard to the opinion of the majority of observers at the present time, the condition is described under the heading "Affections of the Chorion" (*infra*). *See also* "PUERPERIUM—Pathology."

AFFECTIONS OF THE CHORION

Vesicular or Hydatidiform Degeneration (*Hydatid Mole*).—In this condition there is a rapid increase of the connective tissue cells in the stroma or core of each chorionic villus affected. This occurs not continuously, but at intervals, so that a series of swellings is formed along the stem and branches of the villus. The connective tissue forming these swellings then undergoes myxomatous degeneration, the central cells are separated by clear fluid, and later disappear, each swelling thus becoming a small cyst whose wall consists of a thin layer of connective tissue covered by the chorionic epithelium. Concurrently with the cyst-forming degeneration of the mesoblastic cores of the villi, there is also great activity on the part of their epithelial covering, large masses of proliferating syncytium being seen in all sections of hydatid moles. This overgrowth of fœtal epithelium, with its phagocytic action on the maternal tissues, accounts for the unusual depth to which the diseased villi penetrate the uterine wall, and the unusual firmness with which the degenerated chorion is attached. The decidua is usually thickened, some of the cysts being completely embedded within it, while others may invade and even completely perforate the muscular wall of the uterus. The characteristic "hydatid" appearance of the degenerated chorion when expelled follows from the transformation of each villus into a branching string of cysts, varying in size from that of a pin's head to that of a gooseberry.

The change is generally so early in date as to affect the whole chorion, causing the death and complete absorption of the embryo, and the transformation of the whole ovum into a mass of cysts. In rarer cases the degeneration begins later and does not affect the whole chorion. In these instances the remains of the fœtus may be found. In a few cases in which a living child has been born at term, vesicular degeneration has been observed in portions of the placenta.

The frequency of hydatid mole is estimated by Norris and Dickinson at 1 in 20,000 pregnancies. There were 10 cases in 24,500 pregnancies observed at St. Bartholomew's Hospital. Other authors place its occurrence at 1 in 2000 cases. The condition may complicate pregnancy at any age, and cannot be proved to be more frequent in advanced life. It has been said to be rare in primiparæ, and that it tends to follow a series of rapidly recurring pregnancies, but these statements are not supported by facts. It sometimes occurs twice or even three times in the same patient. For this reason, amongst others, the disease is held by some to be of maternal origin, and this view is probably correct, especially as a living child has occasionally been born with part of its placenta in a state of vesicular degeneration. An abnormal condition of the decidua probably permits of overgrowth and subsequent degeneration of the chorion, which in turn kills the fœtus, unless the change begins late and affects only a limited portion of the decidua. It is worthy of note that about half the recorded cases of chorioma malignum have occurred after hydatid degeneration of the chorion, a fact which suggests that the two conditions have a common cause. According to the author's observations, the decidual cells normally exercise a phagocytic action upon the fœtal epithelium, checking undue invasion of the uterine wall by the chorionic villi. If this view is correct, any disease of the decidua which prevented the development of the decidual cells or interfered with their phagocytic action would admit of abnormal growth and activity on the part of the chorionic villi, and would thus afford a primary factor in the causation of both vesicular degeneration of the chorion and of the so-called chorioma malignum, if we suppose that this new growth is indeed composed of fœtal elements.

In the early weeks of pregnancy nothing abnormal is observed. Morning sickness is frequent, the breasts enlarge and are tender. The cervix softens at the usual time, and purple discoloration of the vagina is observed in most cases. Amenorrhœa is complete, as a rule, for some months; but irregular discharges of blood may occur for some time before the hydatid mole is expelled. The fœtal heart may be heard in cases in which the onset of the degenerative process is late, and uterine souffle is occasionally observed; but both these signs are absent as a rule. Uterine enlargement is abnormal, in most cases being much more rapid than in normal pregnancy, while the organ has a softer and more "boggy" consistency than usual. In certain cases, however, the ovum is cut off from the uterine wall by extravasated blood, but is retained in the uterus. Shrinking then occurs, and the uterus remains smaller than it would be at a corresponding period of normal pregnancy, just as in the case when a fleshy mole is retained. In such cases the uterus is firmer to the feel than usual. The rhythmical contractions which characterise the gravid uterus are almost always absent. The attention of the patient is generally attracted by irregular hæmorrhages and the escape of watery blood-stained discharge, which sometimes contains vesicles detached from the chorion, and has then been described as resembling red currant juice with white currants floating in it. The mass is generally expelled before full time

is reached, its separation being rendered difficult by the deep penetration of the degenerated villi into the maternal tissues, and their consequent firm attachment. The same circumstances often cause the expulsion of the mole, to be accompanied by severe hæmorrhage, and also favour the occurrence of septic infection and of subsequent subinvolution.

The diagnosis depends upon the recognition of the symptoms and signs of pregnancy followed by the disappearance of these, with the exception of enlargement of the uterus. The growth of the uterus must then be observed, and if it is not proportionate to the advance of time the presence of vesicular degeneration must be suspected. The occurrence of irregular bleeding confirms the suspicion, which may be rendered a certainty by the appearance of vesicles in the discharges. In the absence of a clear history and of vesicles in the discharge, there is difficulty in distinguishing the condition from accidental hæmorrhage (*see* page 287), and it has also been confused with placenta prævia and with hydramnios. It is impossible to distinguish from fleshy mole a hydatid mole which has been retained dead in utero until shrunken and firm. True hydatid cysts have occasionally discharged their contents through the uterus or vagina, but in such cases a careful examination of the structures discharged at once reveals their true nature.

The prognosis in vesicular degeneration of the chorion is always grave, as deaths from hæmorrhage, anæmia, septicæmia, and pyæmia are frequently recorded. These, as well as perforation of the uterus, are favoured by the deep penetration of the uterine wall and the consequent firm attachment of the mole. Dorland estimates the mortality at 18 per cent. The prognosis must be considered still more grave in view of the fact that more than half the recorded cases of deciduoma or chorioma malignum have followed vesicular degeneration of the chorion.

The uterus should be emptied as soon as the diagnosis is made, or before this can be done if bleeding is severe. If dilatation of the cervix has commenced, packing the vagina with gauze, or distending it with a rubber bag, may be sufficient to ensure expulsion of the mass. Otherwise tents may be used, with strict antiseptic precautions. It is important to secure complete dilatation, uterine contraction, and natural expulsion of the mass when possible. In any case, it is necessary to ascertain by digital examination that the uterine cavity is thoroughly clear, carefully avoiding injury to the uterine wall, and thereafter to take every precaution against septic infection and against subinvolution.

Myxoma Fibrosum.—This name was applied by Virchow to a rare form of degeneration of the chorion, which results in the production of solid masses of fibro-myxomatous tissue instead of the more familiar cysts. Proliferation of the connective tissue cells of the cores of the villi is followed by some degree of fibrous change, the villi forming solid rods. The epithelium remains for the most part unchanged. This condition may be seen in the same specimen, together with cystic degeneration ; and it may also occur in portions of an otherwise healthy placenta, as happened in Virchow's case.

Berry Hart has described a case of "fibrous mole," which was probably due to prolonged retention *in utero* of an ovum which had undergone fibro-myxomatous degeneration. A patient had a threatening of abortion at the third month, and thereafter had slight occasional hæmorrhages during nine months, at the end of which she expelled a pear-shaped mass measuring $4\frac{1}{2}$ by $2\frac{1}{2}$ inches. On section this was seen to be composed almost entirely of a white glistening mass, coarsely granular, with a small amniotic cavity at the lower end, but no fœtus. On microscopic examination the mass was seen to be made up of hypertrophied villi, the cores of which were made up of wavy fibrous tissue, with very few corpuscular elements, and hardly any vessels. Where the villi were not in contact with one another, their epithelium was fairly well preserved, but at other places the villi, intervillous spaces, and decidual cells were blended, the fœtal epithelium being lost and the decidual cells well preserved. The intervillous spaces contained fibrin.

Epithelioma of the Chorion, or Chorioma Malignum (*Syn.:* Syncytioma malignum).—The authors who have described cases of malignant disease under the above names consider the neoplasm in question to be composed of fœtal epiblast, that is, of chorionic epithelium, which, they hold, invades the uterine tissues, and continuing to grow within the maternal organism, produces a new growth with metastatic deposits. For this reason, a description of the condition must be included amongst those of affections of the chorion. It has been mentioned above that other authorities consider similar, if not identical, new growths to be sarcomata of maternal origin.

Clinical Features.—Parturition (in a few cases), abortion, or expulsion of a hydatid mole (in more than half the recorded cases) is followed by profuse intermittent hæmorrhages and enlargement of the uterus, which usually begin a few weeks after the emptying of the uterus. In a case recorded by Whitridge Williams, bleeding was absent, the appearance of metastases first calling attention to the condition. In those of Runge, Löhlein, and others, the bleeding was delayed for some months. The hæmorrhage causes profound anæmia, and putrid discharge of a watery character follows septic infection of the growth. Fever and cachexia supervene,

metastases appear in the vagina, cervix, and pelvic organs, and also in the lungs, liver, brain, kidneys, and other viscera. Death is usually caused by septicæmia, or by pulmonary or cerebral complications due to metastasis, and occurs as a rule within six or seven months of the beginning of the disease, which is thus the most rapidly fatal of all known malignant growths.

The uterus generally contains irregular masses of new growth of very soft friable consistency, composed largely of blood-clot and necrotic tissue, and varying in colour from greyish red to dark purple. The growth involves the muscular wall of the uterus, and sections of it show, besides blood and debris, cell elements of two kinds, and occasionally complete chorionic villi. The cell elements are (1) large individual cells, round, oval, polygonal, or spindle shaped, with large nuclei and clearly defined protoplasm; and (2) irregularly shaped masses of vacuolated material containing numerous nuclei, but showing no cell outlines. The metastases in other parts of the body present the same appearances. The spread of the disease is mainly by the blood stream. Schmorl reported a case which he considered to be of this nature, in which the uterus was not affected; and Davis and Harris have described a case in which abortion was induced for pernicious vomiting, the uterus being curetted, and being then healthy. The patient soon died of general carcinomatosis, which the authors consider to be chorioma malignum, although the uterus was found perfectly healthy at the autopsy.

It will be remembered that the fœtal epiblast, as observed in the epithelium of the young chorion and its villi, consists of two layers, which, though not often so distinct in nature as in the figures of some authors, are easily recognisable in favourable specimens. These are (1) a layer of nucleated protoplasm known as the syncytium, and (2) a layer of large cells with distinct outlines known as Langhans' layer. These, it is said, are the physiological prototypes of the elements of the so-called chorioma malignum, the irregular plasmodial masses seen in that growth being supposed to be derived from the syncytium, while the individual cells are the pathological derivatives of the cells of Langhans' layer. The appearances presented by the chorionic epithelium in hydatid mole are said to present a transition stage between the normal chorion and the malignant new growth. In the cases of Schmorl and of Harris and Davis above mentioned the fœtal epiblastic structures are said to have passed through the uterine wall and entered the maternal circulation, filling the maternal organism with deposits of malignant new growth, yet leaving the uterus itself perfectly healthy. Whether these two cases are accepted or not, the fact remains that the chorionic origin of the majority of the recorded cases is believed in at the present time by most writers on the subject.

The diagnosis is easy. Whenever pregnancy, more especially molar pregnancy, is followed by repeated bleedings and persistent discharge, the uterus should be examined, and scrapings from its interior should be submitted to microscopic examination. The characteristic appearances of sections at once make clear the nature of the case.

The prognosis is absolutely bad, unless surgical treatment is undertaken at an early date. If the uterus is removed, the patient has a very fair chance, recovery having occurred in about one-third of the cases recorded. In most of these the uterus was removed before metastases had appeared. In several cases, however, recovery has followed hysterectomy, although metastases were present, the extravasation of blood around these having apparently destroyed their nutrition and life. The treatment consists in hysterectomy performed as soon as possible after the diagnosis is made, as any waste of time in curetting and other partial measures may be fatal to the patient's chances of recovery.

Affections of the Amnion

The pathology of the amnion is best understood when it is regarded as a serous membrane, liable to variations in its powers of secretion and absorption, and also to inflammations which may cause it to throw out exudates which may be plastic as well as serous.

Hydramnios (*Syn.:* Polyhydramnios, hydrops amnii).—The accumulation within the amniotic cavity of an excessive quantity of liquor amnii may begin at any time during pregnancy. If slight in degree, it causes no symptoms, and may pass unnoticed even during labour. If more pronounced, it may attract attention and demand treatment many weeks before term. At the end of pregnancy the quantity of liquor amnii usually varies between one and two pints, but the condition is seldom noticed unless two quarts or more are present. Quantities of fluid such as six gallons have been removed from the uterus at term. If the accumulation is rapid instead of gradual, the condition is known as acute hydramnios, and presents a much more striking clinical picture than the more familiar variety.

The fluid may be derived from the serum of the maternal blood, as is shown by the occurrence of hydramnios together with other serous effusions. It may also be due to the increased secretion by the amnion of fluid derived from the fœtal blood. This may be caused by any circumstance which raises the blood pressure in the umbilical vein, such as disease of the liver in syphilitic fœtuses, and twists or knots in the cord. Excessive secretion of urine by the fœtus and abnormal perspiration through the fœtal

skin are mentioned by various authors as causes of hydramnios. Inflammation of the amnion has also been regarded as a cause of excessive secretion. Tarnier and other authors hold that deficient absorption of liquor amnii, rather than its excessive production, is the true cause of the condition.

The symptoms and signs of pregnancy appear in regular order, but the uterus enlarges more rapidly than usual. The fœtal heart sounds may be inaudible, and, in extreme cases, it may be impossible to recognise the presence of a fœtus even by ballottement. When the uterus much exceeds its normal size, pressure symptoms are produced, the principal being shortness of breath, cyanosis, and abdominal distress. The more rapidly the distension occurs, the more severe are the symptoms—pain, dyspnœa, cyanosis, and vomiting appearing in quick succession in acute cases. The child is often delicate or diseased, and often presents club-foot, spina bifida, or other deformities. Labour is very often premature. (For the effect of hydramnios upon labour see "Labour, Pathology of," vol. v. p. 239.)

The diagnosis of hydramnios should not be difficult when once the fact of pregnancy is firmly established. Even then it is necessary to use care in distinguishing hydramnios from some other complications of pregnancy. Thus ascites with pregnancy must be recognised by percussion made with the patient lying on the side, when the flank will be resonant as a rule. Cystic tumours with pregnancy will be recognised on palpation as separate swellings, although a large ovarian or parovarian cyst with a small pregnant uterus may afford a veritable diagnostic puzzle. Distension of the bladder during pregnancy is recognisable by the use of the catheter. Twin pregnancy can usually be distinguished from hydramnios by palpation of the fœtal parts. If the fact of pregnancy cannot be clearly established, the diagnosis of hydramnios may, however, be very difficult indeed. The treatment of hydramnios during labour is mentioned elsewhere (*see* "Labour, Pathology of," vol. v. p. 239). If serious symptoms are produced before the child is viable, there need be no hesitation as to rupturing the membranes, as the child is so often deformed, diseased, or dead under these circumstances that the prolongation of pregnancy should not be allowed at any risk to the mother.

Oligohydramnios.—Deficiency of liquor amnii causes certain modifications in the course of labour (*see* "Labour, Pathology of," vol. v. p. 240), and in the very early portion of pregnancy it is said to favour the formation of adhesions between the amnion and various parts of the fœtus.

Inflammation of the amnion, with plastic exudation, is a more definite cause of adhesions between the amnion and the skin of the fœtus which may seriously impair the development of the latter. The adhesive material may become stretched as growth goes on, so forming non-vascular bands which stretch or constrict fœtal structures. Amongst deformities in whose causation inflammation of the amnion plays a part may be mentioned exomphalos, anencephalos, dwarfing of limbs, and intra-uterine amputations. Leopold mentions that the cord may be involved in amniotic adhesions, which may thus cause the death of the fœtus.

Rupture of the amnion may occur, the chorion remaining complete. The membranes may then be separated, and the amnion may be rolled up into bands which may become entangled with the cord, and may so interfere with the fœtal circulation.

Cysts have been found within the substance of the amnion, but these are small in size, and have no effect upon the course of pregnancy or of labour.

Diseases of Placenta and Cord

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The subject of the diseases of the placenta has hitherto obtained very little systematic attention. The normal structure of the organ at different stages of development has only in recent years been clearly defined, and the systematic study of its pathology has therefore been hardly practicable. Many of the earlier accounts of pathological conditions must be rejected, because such conditions are now known to result, not from disease, but from the normal processes of maturation and senescence. A short account of these normal processes will therefore rightly precede the consideration of the diseases of the placenta.

Changes due to Ripening. — Certain well-defined changes occur in all healthy placentæ during the last two or three months of gestation. In their nature they are uniform, but in their extent they present great divergences in different cases, divergences which cannot at

present be adequately explained. Stated briefly these changes consist in the occurrence of obliterating endarteritis in a variable number of the middle-sized branches of the fœtal arteries, and in widespread changes resulting therefrom in the affected areas, the most important of which are the formation of numerous solid patches in the spongy placental substance, termed *infarcts*, and the deposition of large quantities of fibrin from the maternal blood upon the villi in the affected parts. Necrosis rapidly destroys all the structural characters of these villi.

In this manner the maternal and fœtal circulations become obliterated in many small areas of the placenta, and numerous groups of villi are rendered functionless. Well-marked changes of this character can be detected in practically all ripe, healthy placentæ, but the amount of placental tissue thus destroyed varies greatly. The marginal cotyledons are usually much more affected than the central ones. Degenerative changes also occur in the serotina during the same period.

These conditions have frequently been erroneously attributed to placentitis, to hæmorrhage, to syphilis, to phthisis, etc., etc.; they are, in reality, due to old age, and are probably preparatory to the normal process of shedding during labour. The young placenta is certainly less easily separated from the uterine wall than the fully ripened organ, and the presence or absence of the changes described probably explains this difference. It is possible also that these changes determine the onset of labour by gradually rendering the placenta inadequate to the further nutrition of the fœtus.

Post-mortem Changes (retained Placenta).—When the fœtus dies, and the ovum is not at once discharged from the uterus, certain changes occur in the placenta as the result of the suspension of the circulation through it. The fœtal circulation is, of course, at once suspended; the maternal circulation through the intervillous spaces fails gradually; fresh blood may be found between the villi here and there in placentæ which have been retained for several weeks after the death of the fœtus. The villi are nourished directly by the maternal blood which surrounds them, and where the maternal circulation has remained active they show no degenerative or necrotic changes. Where the circulation fails the intervillous spaces become obliterated by coagulation of the blood they contain, and the villi in those parts rapidly necrose. The changes they undergo are similar to those found in infarcts in living placentæ, except that obliterative endarteritis is not present; fatty degeneration is very marked in these villi.

A great deal of the pathological work which has been done with the placenta has been undertaken in the case of retained placentæ. Insufficient acquaintance with the normal post-mortem appearances has led to these changes being described as the cause of the death of the fœtus, whereas in reality they have resulted from it. In this manner a great deal of confusion has been introduced into the subject.

Putrefaction.—Dead placental tissue when retained in the uterus is especially liable to the attacks of putrefactive organisms. This is a very common occurrence in the case of portions of placental tissue retained after labour or abortion, and the clinical results are sufficiently familiar to require no notice in this place. When the entire ovum is retained putrefaction is extremely rare. The reason is that the retention cavity is an aseptic chamber, and unless organisms are introduced by operative interference, or make their way in from bowel adhesions or otherwise, the ovum undergoes non-putrefactive necrosis. Cases are, however, recorded where a fœtus has been discharged piecemeal from the uterus, after being retained for many months, or even for years, without mechanical interference. Putrefaction is much more common in the case of an extra-uterine than a uterine ovum, owing to the liability of the former to injury from rupture, etc.

The tissues of the placenta, though prone to certain forms of degeneration, are not subject to such varied or well-defined diseases as most of the adult organs of the body. Perhaps the explanation of this lies in the embryonic character of the tissues themselves, which is preserved throughout the whole duration of the existence of this organ. Fœtal tissues, for example, never show an active inflammatory reaction; they appear to perish under the influence of an irritant which would be insufficient to destroy adult tissues, but would induce in them an inflammatory reaction. The only inflammatory lesion the existence of which in the placenta has been clearly shown is peri- and end-arteritis, which occurs freely in the mature placenta; here, of course, the inflammatory process is of an inactive kind. Lesions formerly described as due to "placentitis" are now recognised as resulting from degenerative processes. The effect of placental lesions upon the life or development of the fœtus appears to depend more upon their extent than upon their nature. A considerable diminution in the total amount of healthy placental tissue appears to be required to influence the fœtus unfavourably. The analogy of pulmonary diseases is not inapt. It is well known that a considerable area of lung tissue may be destroyed or rendered functionless without much embarrassment of the respiratory process. And similarly an amount of placental tissue much less than that provided by nature appears to be quite sufficient to carry on the processes of fœtal nutrition and excretion.

Anomalies of the Placenta.—*Anomalies of*

Size and Shape.—While subject to considerable variations within certain limits, the size of the placenta corresponds during the last three months of gestation to about one-fifth of the total area of the internal surface of the uterus. A marked increase in the area or thickness is usually due to œdema or to some form of degeneration, and thus becomes a pathological condition, not an anomaly.

A few rare instances have, however, been recorded in which the placenta was equally disposed around the entire ovum, instead of being developed at one or other pole. This condition has received the name of *placenta membranacea*. It is, of course, due to persistence of the arrangement invariably found in the first two months of gestation in which the chorionic villi are equally developed all over that membrane. In some mammals (*e.g.* the cetacea) the diffused form of placenta is always met with, and its occasional occurrence in the human species is extremely interesting. A case has also been comparatively recently reported in which the placenta formed a ring around the ovum, indistinctly divided into three lobes. This corresponds to the zonary placenta of some carnivora. It is produced by an anomaly in the distribution of the changes which result in the disappearance of the extra-placental chorionic villi.

Instead of being a single organ, the placenta may be variously divided into lobes, the segments being continuous with one another through membrane over which the branches of the umbilical vessels pass. There may be two lobes (placenta bipartita), either equal or unequal, or three or more lobes may be present (placenta tripartita, p. multiloba). All medical men are familiar with the variety of this condition known as *placenta succenturiata*, in which one or more small outlying portions of placenta, usually circular in shape, are found connected by a bridge of membrane with the main portion of the organ, and are extremely liable to be left behind when the placenta is expelled from the uterus.

Anomalies in the insertion of the cord are also fairly common. Usually the attachment is in the centre of the placental organ; not infrequently it is markedly eccentric, sometimes even to the extent of being attached to the placental margin (battledore placenta). A rarer variety of insertion is that known as the *velamentous*, in which the cord is inserted into the membranes two or three inches or more outside the placental margin. The vessels bifurcate at the point of insertion, and their divisions run between the amnion and the chorion to the placenta, which they enter in the usual manner. The vessels running between the membranes are liable to injury during labour.

The placenta is usually developed equally around the point on the periphery of the ovum at which the allantois and the ventral stalk reach it. If for any reason the placenta is developed unequally around this spot, eccentric insertion of the cord results; if at a distance from this spot, a velamentous insertion results.

Diseases of the Placenta

Inflammation. — A subject which bulked largely in earlier accounts of the morbid conditions of the placenta was *placentitis*. The changes described were usually those already mentioned as senile or post-mortem changes, and their relation to an inflammatory process was a matter of pure speculation. As a matter of fact, we know nothing of inflammation of the fœtal placenta, apart from gonorrhœal or septic infection, as the direct result of inoculation of the gravid uterus. Infection of the ovum in this manner is, of course, incompatible with its further existence, and the dead tissues undergo rapid putrefaction. The gonococcus has been in two cases detected in early abortions attributable clinically to gonorrhœa, but little or no systematic work has been done at the bacteriology of this subject. It is possible that the condition known as "morbid adhesion of the placenta" is the result of an inflammatory affection of the decidua serotina, but we are not in possession of positive evidence upon the point. The fœtal elements are, in any case, not involved in the process. Certain fairly well-defined forms of inflammation affect the young decidual membrane (decidual endometritis), but an account of this will be found in another place. Fœtal endarteritis has already been referred to.

Hæmorrhage. — The conditions known to obstetricians as placenta prævia and accidental hæmorrhage (*see* p. 287) are instances of hæmorrhage due to separation of the placenta from the uterine wall, and in both cases the bleeding occurs during labour or in the later months of pregnancy. With these conditions we are not here concerned. The subject is further limited by the exclusion of hæmorrhages occurring into the chorion before the formation of the placenta; these are dealt with in another place. We therefore exclude from consideration hæmorrhage into the early ovum, and hæmorrhage from separation of the adult placenta. Hæmorrhage into the substance of the placenta (placental apoplexy) occurs in two distinct forms:—

(*a*) Small interstitial hæmorrhages.

(*b*) Large effusions.

(*a*) *Small interstitial hæmorrhages* are not uncommonly found in the form of circumscribed areas of the cut surface of the placenta, which appear to the naked eye to be almost black, and are easily distinguished from the healthy placental tissue by the purplish colour and reticulated texture of the latter. Usually they lie nearer the maternal than the fœtal surface, but they do not show upon the former surface

when the serotina is intact. There is no uniformity about their size and shape; the largest appear to involve an entire cotyledon; there may be many small ones scattered through an otherwise healthy placenta. Under the microscope the hæmorrhagic areas are seen to consist mainly of blood-clot in which are scattered a few atrophied or distorted villi; the villi are more numerous at the margins than in the centre of the hæmorrhage. It may be objected that such a condition as this is not hæmorrhage, because the blood is still contained in the intervillous spaces and has not escaped from its normal channels. But it differs from mere congestion or engorgement of vessels, inasmuch as the surrounding structures (villi) are disturbed, and in part destroyed, by the process. Except in a technical sense, it is interstitial hæmorrhage. These hæmorrhages do not affect the functional activity of the placenta to an extent sufficient to react unfavourably upon the fœtus. It is quite possible that they are produced during labour by the influence of the uterine contractions upon the maternal circulation through the placenta. A case has been recently reported by Dr. Blacker, in which an extensive hæmorrhage of this kind occurred in a very young placenta, from cardiac disease, without inducing abortion or causing the death of the fœtus.

(*b*) *Large effusions of blood* into the placenta are much rarer than the interstitial hæmorrhages just described. Such effusions are probably produced more rapidly, and therefore break up the placental tissue to a much greater extent than the interstitial ones. In consequence, irregular cavities containing blood-clot are formed in the substance of the placenta, limited in recent cases by compressed and broken villi, and in older cases by layers of fibrin forming a false cyst wall. In some of these cases the hæmorrhage is fœtal from rupture of a branch of the umbilical vein or artery in the placental tissue, and sometimes the cavity communicates with the circular sinus (maternal). After accumulating in the placental substance the blood may burst through the serotina, causing some separation from the uterine wall; such an occurrence might cause external hæmorrhage. The fœtus is born dead when hæmorrhages of this kind exist in the placenta. If abortion does not immediately follow its occurrence the effused blood undergoes the usual changes from clotting, absorption, or partial organisation and degeneration. The causes of large placental hæmorrhages are by no means fully known. In a case observed by the writer the mother suffered from albuminuria during pregnancy, and the portions of the placenta unaffected by the hæmorrhage showed extensive pathological infarction, as is so commonly the case in albuminuria. But the whole subject of placental hæmorrhage still requires a great deal of systematic work.

Œdema.—Œdema of the placenta usually results from disturbances of the fœtal circulation or changes in the fœtal blood, and is therefore associated with general œdema of the fœtus, and due to the same causes as this condition. Cases of maternal dropsy have also been recorded in which the placenta was œdematous: in these cases the cause would appear to be maternal; but more often placental œdema is due to a fœtal cause. Instances may be found of obstruction to the arterial return from the placenta by antenatal closure of the foramen ovale, or thrombosis of the umbilical and hypogastric vessels before birth, in which both the placenta and the fœtus were œdematous. The placenta of acardiac monsters is also frequently œdematous.

Within recent years several cases of fœtal and placental dropsy have been carefully recorded, in which there was present a marked excess of leucocytes in the fœtal blood; in one of these cases, recorded by Liefart, the mother suffered from albuminuria and dropsy, which rapidly cleared up after labour. Possibly in these cases the morbid condition of the fœtal blood was due to the increased toxicity of the maternal blood, which has been shown to exist in the albuminuria of pregnancy. Virchow and Franck have observed that in general dropsy of the fœtus of bovine animals the thoracic duct and mesenteric lymphatics are often absent, but this point does not appear to have been investigated in the human fœtus.

An œdematous placenta is larger and heavier than normal, and undergoes a great diminution in weight when incised and drained of fluid. The normal weight of the organ is 16 ounces; in the case reported by Liefart the œdematous placenta weighed 67 ounces. In colour it is pale, and in consistence unusually friable and soft. Microscopically the villi are found enormously increased in size and closely packed together, so that the size of the intervillous spaces is much reduced, and they therefore contain comparatively little blood. Many of the fœtal vessels are thickened or thrombosed, and the stroma of the villi is poor in cells and contains abundant fluid in its spaces. Frequently marked fatty degeneration of the chronic epithelium is also present.

Usually the fœtus dies *in utero*, although this event is not by any means of invariable occurrence. Œdema most seriously impairs the functional capacity of the placenta, but as it is never idiopathic we cannot tell precisely what effect it exerts upon the nutrition of the fœtus. The morbid conditions associated with it may be sufficient to destroy the fœtus.

Pathological Infarction.—It has already been stated that during the last two months of intrauterine life certain changes referable to senility occur in the fœtal placenta, resulting in the formation of small solid bodies in the spongy placental substance termed "infarcts." Under

conditions of health these are neither large enough nor numerous enough to interfere with the functional capacity of the placenta or the nutrition of the fœtus. Within the last decade it has been shown that changes similar in nature, but much more widespread and occurring much earlier in gestation, are found in association with a well-known disorder of pregnancy, viz. albuminuria. This condition has long been known to cause a heavy fœtal mortality, estimated on recent evidence at 54 per cent, and evidence has now been advanced that the changes in the placenta are the cause of the death of the fœtus.

In a marked example of pathological infarction a considerable proportion ($\frac{1}{3}$ to $\frac{1}{2}$) of the placenta is converted into a firm, yellowish, apparently bloodless tissue, which is seen under the microscope to consist of atrophied chorionic villi with large deposits of fibrin, both elements usually showing marked fatty degeneration. Hæmorrhages may be found in the non-infarcted portions of the placenta. In marked cases of albuminuria pregnancy seldom continues beyond the thirty-sixth week, and the earlier the onset of labour the more marked are the placental changes. As a rule the most extensive changes of all are found in the placentæ of fœtuses which have perished *in utero*, and have been born in a state of maceration. The infarction appears to depend upon an obliterative endarteritis of the fœtal vessels. It has long been known that the fœtus of albuminuric mothers is smaller and lighter than the normal fœtus of the corresponding period of gestation; it is, in fact, under-developed, and possesses but low vitality.

Degenerations.—The following forms of degeneration affect the placenta:—

1. Vesicular degeneration (hydatidiform mole).
2. Myxoma fibrosum.
3. Fatty and calcareous degeneration.
4. Cystic degeneration.

1. *Vesicular degeneration* is a form of myxomatous degeneration of the stroma of the chorionic villi which usually occurs at an early period before the differentiation of the placenta begins; it is therefore more commonly a disease of the chorion than of the placenta, and will be found fully described in that relation in another place. In rarer instances, however, it occurs at a later period, and is then found in the placenta. The disease has the main general characters as in the hydatidiform mole, but the whole placenta is seldom affected; usually one or two cotyledons degenerate, while the rest of the organ escapes.

2. *Myxoma fibrosum* is a form of myxomatous and fibroid degeneration of the chorionic villi which is usually described as a new growth, although it would be more accurately classed as a form of degeneration. It will be described under "Tumours of the Placenta," p. 253.

3. *Fatty and Calcareous Degeneration.*—Fatty degeneration is rarely found in the placenta except, as the result of pre-existing lesions. The observations of Barnes and Druitt, which are still sometimes quoted in textbooks as the first description of primary fatty degeneration, have been shown to be incorrect. A certain amount of fat is contained in the normal human placenta, being located in the plasmodial layer of the chorionic epithelium and in the superficial layers of the decidua. This may be easily distinguished from pathological deposits, as it occurs in minute non-confluent droplets and is very small in amount. A good deal of fat is also found in white infarcts, the older infarcts containing the larger deposits. Fatty degeneration is perhaps most commonly met with as a sequela of pathological infarction in albuminuria, and is probably the direct result of the extensive arterial obstruction of the fœtal vessels which occurs in that condition. It is also not uncommonly found in connection with placental œdema and in placentæ retained *in utero* after the death of the fœtus. There is no evidence of any well-defined relation between syphilis and fatty degeneration, as was so frequently stated in the earlier papers on this subject.

In parts which have undergone fatty degeneration a deposit of lime salts is often found. In a ripe placenta numerous small thin calcareous plates are present upon the maternal surface; they occur in the serotina, and as a rule do not involve the villi. Instances are on record where almost the entire uterine face has been thus covered with a thin calcareous plate. Such deposits do not appear to affect the functional activity of the placenta.

4. *Cystic Degeneration.* — By this term is understood the formation of cysts by a process of degeneration. Cysts occur but rarely in the placenta, being occasionally observed upon the fœtal surface of an otherwise healthy organ. Here they occur as small multiple collections of clear yellowish fluid situated beneath the amnion. This membrane can be readily peeled off the cyst which appears to be in the substance of the sub-amniotic chorion or the basal plate of placental chorion from which the villi spring. Occasionally similar cysts occur in the amnion itself. They are produced by a process of myxomatous degeneration of the connective tissue, and possess no epithelial lining, their walls being formed of degenerating and compressed connective tissue. The placental connective tissue is all of embryonic type, and is very prone to this form of degeneration. These cysts never exceed the size of a pigeon's egg, and do not interfere in any way with the functional activity of the placenta.

Tuberculosis. — Tubercular disease of the placenta is probably rare; at any rate the number of cases recorded in recent years is

exceedingly small. It has been shown to occur, however, in women affected with chronic pulmonary phthisis, or with acute miliary tuberculosis. Cases occurring under both of these conditions have been recorded by Schmorl and Kockel, Lehmann, and Londe.

Placental tubercle possesses the same general characters as tubercle of other organs. The nodules are found in the substance of the cotyledons more often than upon the surface, and are more abundant in the marginal than in the central parts. They are usually firm and fibrous in texture, but sometimes caseating. Under the microscope they are seen to contain characteristic giant cells, and tubercle bacilli can be recognised by the usual differential staining reactions. Inoculation experiments upon rabbits and guinea-pigs show that the bacilli are active. Schmorl describes the tubercles as occupying two distinct positions in his cases; usually they were upon the surface of the villi, encroaching upon or obliterating the adjacent intervillous space. Sometimes, however, they were situated in the stroma of the villus, the surface epithelium covering it being intact.

In almost all the recorded cases the maternal disease was so advanced that the mother died either during labour or very soon afterwards. In the six cases recorded by Schmorl, and Kockel, and Lehmann, the fœtus died soon after birth, and a post-mortem examination was made. In only one case, however, was tubercle found in the fœtal organs, and in this case there were characteristic deposits found in the lungs, liver, spleen, and left kidney, and microscopically the tubercle bacillus was readily recognised. In all the other cases, not only could no tubercle be found in the body of the fœtus, but inoculation experiments were also attended by negative results. The blood from the tubercular placenta was, on the other hand, usually infective. From these facts it would appear that the fœtus is to a certain extent protected from infection from the maternal blood by the placenta, the organisms being detained there as in a filter. In only one of the recorded cases is it stated that the fœtus died *in utero* before the onset of labour.

The diagnosis of placental tubercle is impossible except by the microscope. It does not destroy the fœtus, and gives rise to no symptoms which can be recognised during life.

Tumours of the Placenta.—New growths are among the rarest diseases which occur in the placenta. A Norwegian writer, Alin, succeeded in collecting records of 23 cases from the literature of all countries, and 6 other cases have been since reported, making a total of 29 in all. As reported by the various writers, these tumours numbered, myxo-fibroma (myxoma fibrosum, of Virchow), 14; fibroma, 5; angioma, 5; fibromyoma, 2; sarcoma, 3.

Many of the earlier cases are, however, insufficiently reported, and the nature of the growth remains open to doubt. It will be noticed that all the above are varieties of connective tissue tumours; no new growth arising in epithelium appears to have ever been reported. The malignant uterine growth known as deciduoma malignum cannot be regarded as a tumour of the placenta, even if the current theory of its origin in fœtal epithelial cells be accepted. Tumours of the placenta do not exert such a markedly unfavourable influence upon the fœtus or the mother as might at first be supposed. Out of the 29 cases a living fœtus was born in 25; the remaining 4 were born dead; a fœtal mortality of 1 in 7 cannot be regarded as a high mortality for a serious placental lesion. Labour was not unfavourably influenced in any case. The frequency with which myxo-fibroma occurs in the placenta is very marked in comparison with the other new growths; it is the only one which need be described in detail.

Myxo-fibroma (myxoma fibrosum).—Although the commonest form of placental tumour it is a rare disease. It occurs as a firm, rounded swelling, usually in the substance of the placenta, and forming a protuberance upon both surfaces, or in the case of growths of smaller size, upon the maternal surface only. Less often the tumour forms a pedunculated mass attached to the maternal surface, and has a more or less definite branching arrangement. The fœtal surface of the tumour is covered with the amnion, which may be stripped off it, showing that the membrane is not involved in the tumour. Often there is a distinct fibrous capsule, and occasionally there are multiple growths. The microscopic characters of the growth are not uniform, and are described in very different terms by different writers. In the main it appears to consist of altered villi. The villi are enormously enlarged—upon this point there is practical unanimity—by increase of their stroma. The characters of the altered stroma appear to vary, but speaking generally, the change is one of myxomatous and fibrous new formation, the relative proportions of the myxomatous and fibrous tissues being variable. The chorionic epithelium does not alter, and is often retained upon the enlarged villi. Sometimes the vessels of the affected villi are much enlarged, forming the variety of the growth called *myxoma fibrosum telangiectodes.* The growth is of course benign in all cases, and the parts of the placenta not involved in it are healthy.

Diseases of the Membranes. — *Decidual Endometritis.*—This condition is a disease of the early ovum, affecting the decidua in all parts, and occurring at a period prior to the formation of the placenta. Two distinct forms are described, viz., the *glandular* and the

interstitial. The *glandular* form corresponds to the common glandular form of endometritis in the non-gravid uterus, and probably results from conception occurring in a uterus whose endometrium was thus affected. The decidual membrane is of unusual thickness, and frequently presents small polypoid cysts upon its uterine surface, formed no doubt by cystic dilatation of the deeper portions of the uterine glands. The microscope does not disclose any actual inflammatory changes, the morbid condition being characterised mainly by glandular proliferation, with some increase of the normal inter-glandular stroma. The *interstitial* variety, on the other hand, is a true inflammatory change characterised by round cell infiltration of the whole membrane, congestion of vessels and interstitial hæmorrhages, with inconsiderable changes in the glands. Emanuel has shown that it may be caused by the gonococcus, and has demonstrated the presence of this organism in the inflamed membrane. Cholera and certain of the exanthemata are also said to cause acute hæmorrhagic endometritis in pregnancy, but further observations are required in this direction.

The symptoms associated with decidual endometritis are pain and hæmorrhage during the first three months of pregnancy, and a marked tendency to abortion. If the ovum survives that period atrophy of the decidua begins, and the danger abates. Diagnosis can only be established by microscopic examination of the membrane. It is possible that morbid adhesion of the placenta is a sequela of this condition.

Diseases of the Umbilical Cord.—The umbilical cord is less prone to disease than the placenta; the number of morbid conditions which are known to occur in it is very small indeed.

Such accidental anomalies as undue or diminished length and thickness, true and false knots, and excessive torsion, need only be mentioned; the latter condition is only found in the case of a dead fœtus, and is now usually believed to be a post-mortem occurrence. Syphilitic endarteritis and periphlebitis have been described in the umbilical arteries, sometimes to the extent of causing actual obliteration of the lumen. Degeneration cysts are sometimes met with of the same nature as those occurring in the placenta. A case of myxosarcoma of the cord has been described by Winckel.

Intra-Uterine Diseases and Death of Fœtus

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Physiology.—The study of the intra-uterine diseases of the fœtus is a subject which has been systematised only within recent years. The pathology of ante-natal life may be divided into three parts: the pathology of the germ, of the embryo, and of the fœtus; and it is with the last-named portion that we have here to do. It has more in common with the pathology of post-natal life than have the pathology of the embryo and that of the germ; for it has mainly to do with diseases while these others have to do with monstrosities and malformations and predispositions. Nevertheless the diseases from which the fœtus suffers show many peculiarities, and are not always recognisable as the same maladies as those which are met with in adult life or even in childhood. These peculiarities are due to certain circumstances, such as the intra-uterine environment, to which attention will be drawn immediately; in the meantime it is necessary to have some knowledge of the physiology of fœtal life.

The *physiology of the fœtus* throws much light upon its pathology. Its chief feature is its parasitism. The fœtus spends the whole of its existence, about seven and a half months, in the interior of the uterus: it is immediately surrounded by the liquor amnii, which, serving as a natural water-cushion, protects it from sudden shocks and jars; the uterine walls, by their growth and distensibility, allow increase in size and freedom of movement to the fœtus, while they shield it from harm, and maintain by their vascularity that constant temperature so needful for healthy development; and, external to the uterus, are the partly osseous, partly muscular pelvic and abdominal walls, which serve still further to secure the safety of the tender organism. In its protected position the fœtus makes little call upon several of its organs; its lungs are absolutely quiescent; its stomach, intestine, skin, kidneys, and lymphatic glands are nearly so. The heart and liver, however, are active; the thymus, thyroid, supra-renal glands, and sympathetic system play a considerable part in the physiology of intra-uterine life; and the brain and spinal cord are capable of replying to all the demands made upon them. The most important of the fœtal organs, however, is the placenta, although the fœtus is only part-possessor in it, and it lies

outside the fœtal body. It is the dominating influence in fœtal life, and is absolutely essential to the unborn infant; in fact, fœtal vitality, although not structural integrity, may be maintained by the help of the placenta alone. The study of monstrosities has proved that almost all the organs may be absent, or so malformed as to be useless; if only the placenta be healthy and active the fœtus lives on until its expulsion from the uterus deprives it of the assistance of the placenta, and so brings about its death. Just as post-natal life is ushered in by a transition period, the neo-natal, during which the organism is being readjusted to its new environment, so fœtal life begins with a period which may be called neo-fœtal, in which the circumstances which prevailed during embryonic life are being replaced by those which characterise fœtal existence; the outstanding phenomenon of this transition time is the establishment of the placental connection. Embryonic life has as its most striking feature evolution or development; it is a period of planning, of arranging; and in it the apparently similar cells of the early embryo are grouped together to form the various organs of the fœtus. The chief result of physiological activity on the part of the fœtus, on the other hand, is rapid growth along the lines which have been already laid down. At the same time the whole organism does not pass at the same moment out of the embryonic into the fœtal period; there is a projection of the embryonic into the fœtal in the physiology of ante-natal life.

The *pathology of the fœtus* is characterised by diseases as distinguished from embryonic pathology, which has malformations and monstrosities as its peculiar possession. Further, it is probable that with certain inconsiderable exceptions the fœtus is liable to the same diseases as is the adult or child. In a broad sense it may be said that the pathology of the fœtus is co-extensive with that of the adult. The fœtus enjoys a partial immunity from the attacks of parasites which produce skin diseases in extra-uterine life, and it is to some extent protected from external violence by its environment; with these exceptions, it has the same wide pathological possibilities as has the adult or child. It is true that elaborate details concerning the mode of onset, symptoms, and progress of fœtal maladies are not to be found in textbooks of medicine; but this is due to the fact that these diseases are during their evolution hidden from the eye in the darkness of the womb, and only come under the observation of the medical man when the expulsion of the fœtus from the uterus happens to coincide with the stage of full development of the disease, and occurs before the death of the fœtus has taken place. There are other reasons, also, of a more commonplace kind which help to explain how it is that more is not known about these intra-uterine maladies: these are the low estimate which has been placed upon the value of fœtal life, the absence of exact knowledge regarding intra-uterine physiology, and the invasion of the subject by such unscientific imaginings as those connected with maternal impressions. The peculiarities of fœtal diseases also mask them and prevent their identification with the diseases of the adult.

The *classification* of fœtal diseases and morbid states may be founded upon their pathology, their distribution, or upon their etiology; but no satisfactory scheme has been elaborated, nor is one possible until our knowledge of the whole subject is much enlarged. In the meantime it is convenient to divide the morbid states from which the fœtus may suffer into (1) idiopathic diseases, (2) transmitted diseases, (3) traumatic morbid states, (4) transmitted toxicological states, and (5) pathological conditions of the fœtal annexa, the placenta, membranes, and cord. A special department has to do with fœtal death and its pathology.

The *causes of the peculiarities of fœtal diseases* are to be looked for in the influence of the intra-uterine environment, in the placental factor, and in the embryonic factor. A few words must be said in explanation of these various modifying agencies. The special conditions which characterise intra-uterine existence are responsible for several of the peculiarities which are impressed upon the maladies of this period of life. Traumatism, for instance, as it is understood in post-natal life, is a rare occurrence in fœtal life. So well is the unborn infant protected that it is very uncommon for it to be the subject of fractures, dislocations, and wounds apart from those received during the act of birth. Of course now and again such injuries are met with as the result of gross maternal traumatism, such as abdominal compression and laceration; but they are rare, and the fractures and so-called intra-uterine amputations which are recorded have probably another origin (from amniotic pressure) to which reference will be made in the proper place. The modifying effect of environment is seen also in the characters which some of the exanthemata take on when they occur *in utero*. Thus, in fœtal smallpox the eruption on the skin resembles that which occurs on the mucous surfaces in later life, a fact which is probably due to the influence which the circumambient liquor amnii exerts; there is rarely a noteworthy formation of crusts, and the resulting cicatrices are very slightly marked; and the pustules do not appear to affect the face to the same extent as they do in the smallpox of later life, probably because the face is not less protected from the light than are the other regions of the body. Possibly the intra-uterine surroundings have also brought it about that erysipelas, at any rate in its cutaneous manifestations, is very rare in the fœtus. The enormous thickening of the epidermis in fœtal ichthyosis has been

ascribed to the comparative absence of friction *in utero*. The peculiarities of the changes which follow fœtal death must certainly be largely due to the environment of asepsis with which the unborn infant is in normal circumstances surrounded. The placenta, which plays so important a part in the physiology of the fœtus, has an equally important effect upon its pathological developments. Thus, the presence of the placenta makes it possible for the fœtus to be diseased in structure to a very advanced degree without the suspension of its vitality. An enormous amount of morbid change may be present without the cutting short of intra-uterine life. So long, for instance, as the fœtal malady attacks organs whose functions are performed in whole or even in part by the placenta, the induced morbidity is only potential. The lungs may be solid from pneumonia and yet no inconvenience be caused to the fœtus *quâ* the state of its lungs so long as the placental economy is maintained. This potential morbidity becomes real at birth. The potential mortality of the fœtus is another effect of the placental predominance. An amount of structural change quite incompatible with extra-uterine existence may be present *in utero* without causing fœtal death. A fœtus with general dropsy may come to the full term with its peritoneal, pleural, and pericardial cavities loaded with fluid, and with advanced changes in its internal organs; it may even show signs of life at birth; yet it invariably dies in a few minutes or at most hours thereafter. The potential mortality became real at birth. It is in virtue of this effect of the placenta that grossly malformed embryos are carried through the fœtal period of intra-uterine life and brought to the light; perhaps the mother is thus saved from the dangers of intra-uterine death or premature delivery. Again, the placenta acts sometimes, at least, as a barrier preventing the access of poisons and toxines circulating in the maternal system to the fœtal economy. It has been shown that in the lower animals, at any rate, the placenta has a real affinity for some toxic bodies, and by storing them up in its substance prevents them passing to the fœtus. In this respect it resembles the action of the liver in the adult, being an accumulator of poisons. Of course it does not necessarily prevent their passage, although it may store them up. With regard to the effect of the placenta in keeping back microbes from the fœtus there is still much uncertainty. The idea that it acts always as a filter must be abandoned; that it acts often as a prophylactic barrier is also open to grave doubt. Bacteriological researches have shown that through it can pass the bacilli of anthrax, of fowl cholera, and of typhoid fever, the pneumoccus, the streptococcus, the spirillum Obermeyeri, the bacillus of glanders, the pathogenic organism of hydrophobia, and perhaps the hæmatozoon of malaria. So we have now to look at the opposite side of the question, viz. the placenta as a means of access of germs and toxines to the fœtus. It is certainly the chief if not the only avenue of entrance to the fœtal economy; for it cannot be accepted that substances often pass by the membranes into the liquor amnii, and are then swallowed and so reach the interior of the unborn infant. It would seem also that it is not necessary for the placenta to be macroscopically diseased, as by hæmorrhage into its substance, in order that the organisms may pass; in fact, in some cases in which the germs have passed, the placental tissue does not appear to have been even microscopically affected. It cannot be said that the conditions which increase or diminish placental permeability are yet well known; but one or two important conclusions have been arrived at. It is, for instance, apparent from the study of the comparative anatomy of the placenta that its permeability must vary in different species with the thickness of tissue intervening between the maternal and fœtal circulations; in this respect it would appear, from the extensive researches of Duval, that the human placenta occupies an intermediate position between that of the rodents and that of the ruminants. Again, there is reason to believe that placental permeability varies with the epoch of pregnancy reached. It would seem also, although the evidence rests on experiments on animals and not on observations on the human subject, that the presence in the blood of toxines, as tuberculine, or of alcohol, increased the permeability of the placenta, while the previous inoculation of the system with an antiseptic, such as corrosive sublimate, made it more difficult for the germs to pass. The fact that it is through the placenta that infection reaches the fœtus has an important bearing upon the position of primary lesions in the fœtus. The germs and toxines arrive at the body in the blood, passing first through the placenta and umbilical vein, then either through the liver or ductus venosus, then through the heart, and so to the whole circulation. It is no matter for wonder, therefore, that the primary pathological changes are commonly found in the liver or heart, or placenta itself. Congenital tuberculosis is not often met with, but when it has been demonstrated it has been noted that the lesions were inconsiderable in the lungs and very extensive in the liver and spleen. Again, erysipelas in the mother has been found associated with streptococcic endocarditis in the fœtus, and not with erysipelatous inflammation of the fœtal skin. The placenta has been already referred to as the most important organ of the fœtal economy; for this very reason morbid changes in it are very immediately prejudicial to the

fœtus. The healthy placenta may enable a diseased fœtus long to survive, but when it is itself the seat of disease it is very often the cause of fœtal death or of premature delivery. In order, therefore, to arrive at the cause of intra-uterine death it is very important to have the placenta for examination. While the placenta and the intra-uterine environment serve to account for some of the peculiarities of fœtal maladies they leave unexplained not a few. Many of these are due to the projection into fœtal pathology of the results of embryonic pathology. There is good reason for believing that malformations and monstrosities are the product of morbid agencies acting on the organism during the embryonic period, and the fœtus may therefore come into its epoch of intra-uterine existence with pathological states in it which are really embryonic. These must necessarily modify the morbid conditions which are superposed during the fœtal period, and fœtal diseases are affected in their manifestations by pre-existing malformations. Further, it is more than probable that the malformations themselves are altered in some respects by the diseases which supervene. It is, therefore, not to be wondered at that fœtal maladies have certain peculiarities which serve to distinguish them from the diseases of post-natal life.

The *diagnosis* of fœtal diseases while the fœtus affected by them is still *in utero* is an exceedingly difficult matter; even the Röntgen rays have not as yet done much to pierce the obscurity which surrounds fœtal morbid processes, for the ossification of the unborn infant is incomplete, and the mother's pelvis throws a dark shadow. Some little progress has, however, been made in this exceedingly difficult field of clinical investigation. Since the infant is in close relation to the chemical and vital processes going on in the body of its mother, a full consideration of all these processes, as they show themselves in the general maternal health, cannot fail to throw some light upon the general fœtal health. The better the state of health of both parents the more likely it will be that the progeny will pass through intra-uterine life without illnesses. It is necessary, therefore, in the first place, to take into account the past history of the parents; especially is it important to investigate the past obstetrical history of the mother, for fœtal diseases often show a curious tendency to repeat themselves in consecutive pregnancies. This repeating tendency has been noted in general fœtal dropsy and in fœtal ichthyosis, and it is, of course, evident in fœtal syphilis. It may be mentioned in passing that the same phenomenon has been remarked in teratology; women, who may in consequence be called monstriparæ, having been known to give birth to several monstrosities in succession. The influence of the father sometimes makes itself felt, for cases are on record in which a woman has had healthy children by a first husband, and dropsical or ichthyotic ones by a second, her own health having continued apparently unaltered. It is necessary also to inquire into the symptomatology of pregnancy, for abnormal intra-uterine conditions sometimes give evidence of their presence by alterations in the normal manifestations of gestation. Unusually active or unusually inactive fœtal movements may occur in fœtal disease, and I have the notes of a case of fœtal singultus diagnosed by the peculiarity of the movements made by the infant still *in utero*. All matters, such as the exposure of the mother to infection or to traumatism during pregnancy, ought to be noted. The fact that she herself is suffering from malaria, smallpox, measles, scarlet fever, typhoid, and the like, is strong presumptive evidence that her infant *in utero* is similarly affected. In cases of general tuberculosis of an advanced type, it is now known to be possible for the fœtus also to be tubercular. The actual examination of the mother, and especially of her abdomen, may give some hint as to the state of the unborn infant. Thus the presence of hydramnios has been found to be very constantly associated with an intra-uterine pathological state, either of the nature of a disease or a monstrosity, and hydramnios can generally be diagnosed by careful examination. Again, it has been noted that occasionally the fœtal heart sounds have been impure, and the diagnosis of fœtal heart disease or malformation has on several occasions been made and confirmed after the birth of the child. During labour it is sometimes possible to diagnose a fœtal disease, and even then the diagnosis is not without value, for it enables the medical man to forewarn the interested parties of the state of matters, and so save himself in some measure from the reproach attaching to the unexpected advent of a diseased infant. The diagnosis of intra-uterine death will be considered later.

Intra-uterine therapeutics is a subject which has as yet made little progress, and it can hardly be expected to make much until intra-uterine diagnosis has been perfected. Nevertheless, in some few directions the outlook is in some degree hopeful. The study of fœtal pathology has shown that it is dangerous for the pregnant woman to expose herself to the infection of the exanthemata, even if she herself have suffered previously from the special fever in question, for her acquired immunity does not extend to her unborn infant. In the presence of an epidemic of variola, the vaccination of the pregnant woman becomes a rightful precaution, not so much for her own sake as for that of her fœtus, for it has been shown that sometimes the latter is thus rendered immune to subsequent vaccination, and therefore probably to smallpox. In the same way quinine may be given to the pregnant woman sur-

rounded with the malarial poison. The giving of mercury to the syphilitic mother for her own sake, but also for that of her unborn infant, need not be specially referred to, for it is an everyday matter in practice; but it is well to remember that when the father alone is affected, it is still right to administer mercury to the mother when pregnant for the purpose of directly acting on the child. Chlorate of potash is a drug which has a good effect upon certain abnormal intra-uterine states, such as the tendency to habitual abortion or premature labour; possibly it proves beneficial by its action on the placenta; it may be combined with iron with advantage. There are certain cases in which, on the other hand, it may be good treatment to induce labour before the full time, in order to save the infant from a malady which has been shown to come on about the last month of intra-uterine life, and the same course has been taken when a woman has on previous occasions given birth to infants which die in labour simply on account of their post-mature development.

Special Diseases of the Fœtus

The individual diseases of the fœtus cannot all be considered in this article, for that would entail the writing of a system of antenatal medicine; it will suffice if the maladies whose pathology differs most markedly from that of post-natal life be dealt with. The first that call for description are the idiopathic diseases of the skin.

Skin Affections. — *Fœtal ichthyosis* (*major degree*) is one of the most deforming of all the congenital skin affections. It is histologically a hyperkeratosis, and is characterised by the existence all over the body of horny epidermic plates, separated from each other by narrow fissures or deep and broad grooves; this gives to the infant the appearance which has earned for him the name of "harlequin fœtus." The mouth, eyes, and anus show ectropion, and the nose, ears, and limbs are more or less stunted in their growth from the compression exercised upon them by the thickened epidermis. The disease has shown family prevalence, that is to say, several infants of the same parents have been affected with it. It is not compatible with extra-uterine life, for in all the recorded cases death has followed birth within a few hours, or at most days. Lactation is always much interfered with by the state of the mouth. The horny layer of the epidermis is enormously thickened; this is the most characteristic lesion, and justifies the name ichthyosis, although in its adult form ichthyosis never approaches in severity to the fœtal.

Fœtal ichthyosis (*minor degree*) is characterised by the birth of an infant which appears as if it were coated all over with a layer of collodion. In a short time desquamation begins, and may go on more or less continuously for years. There is not usually much ectropion of the eyes and mouth, but cases have been reported showing the intermediate degrees between this, the benign, and the more grave type of fœtal ichthyosis which has been described above. It is not incompatible with a prolonged extra-uterine existence, and is capable of showing improvement under suitable treatment by warm alkaline baths and inunction with lanoline or glycerine.

Ichthyosis herpetiformis is characterised by the presence of papillomatous growths, distributed along the line of one or more of the cutaneous nerves, usually limited very exactly to one side of the body, and more or less pigmented. To this condition many names have been given, such as nervenævus, nævus neuroticus unius lateris, ichthyosis linearis neuropathica, and papilloma neuroticum; and it may be regarded as a localised form of congenital ichthyosis hystrix. It has been ascribed to fœtal herpes zoster. Division of the nerve supplying the affected area has been followed by cure.

Tylosis palmæ et plantæ is another form of hyperkeratosis, but it is limited in distribution to the palms of the hands and soles of the feet. It is very markedly hereditary. The lesion exists at birth, but only becomes evident when pressure on the parts becomes considerable. It has been also called ichthyosis palmaris et plantaris and keratoma plantare et palmare. Improvement may be obtained by painting the surface with a solution of salicylic acid in ether (5 per cent).

Fœtal keratolysis, or desquamation of the cuticle, is a normal phenomenon at the end of the first week of life, but it is exceptional to find it in the infant at the moment of birth. When it is met with in the fœtus it may signify fœtal measles, scarlatina, erysipelas, syphilis, anasarca, or simply post-maturity of the infant. Antenatal desquamation of the cuticle has by some been regarded as a certain sign of fœtal death, but it is undoubtedly found occasionally in the living infant.

Congenital hypertrichosis, or abnormal hairyness, may be general or local. In the former there is a persistence of the general lanugo-covering of middle fœtal life, and individuals thus affected are known as "hairy men," "missing links," and "hommes-chiens." There is sometimes defective development of the teeth. Localised hirsuties has its commonest form in the well-known *hairy nævus or mole*. It occurs on the face, back, or limbs, and may be associated with defects of the skeleton, such as spina bifida occulta and encephalocele. It is often ascribed to the so-called maternal impressions. The condition is of importance only on account of the deformity which it causes, and successful treatment by means of electricity or the Röntgen rays may be obtained.

Congenital alopecia, or *hypotrichosis*, may also be general or local; the former is commonly hereditary, and the latter is due to causes which are not at all well known, but which have sometimes been thought to include the antenatal spontaneous cure of a vascular nævus. Dental defects may be associated.

Pigmentary anomalies of the skin are sometimes met with at birth. Among these chromatoses must be reckoned the well-known *nævus pigmentosus* and the rare general *melanoderma*, in both of which there is an excess of pigment in the cutaneous tissues. The pigment may, on the other hand, be *absent* either in restricted areas of the body, as in the white streaks (sometimes unilateral) seen sometimes in dark-skinned peoples, or as a general affection in albinism.

Congenital pemphigus generally points to syphilis, but may sometimes be due apparently to a prenatal abnormal tendency to the formation of bullæ quite apart from the infection with syphilis. During birth the bullæ are commonly ruptured and the epidermis removed, producing the appearance of bare areas.

Congenital absence of the skin and *congenital ulcers* have quite a different mode of origin. They probably occur when there have been adhesions between the amnion and the fœtal skin (sometimes of a tubular nature), and these being torn through in the act of parturition, areas of denuded skin are left to mark the site of the amniotic union. They are of some medico-legal importance, as they may simulate the results of traumatism.

Fœtal Bone Diseases are very difficult to reduce to the terminology of adult osseous lesions, for the reason that before birth the skeleton is not yet fully developed. This fact in part accounts for the multiplicity of names which have been given to the fœtal maladies of bone, such as chondrodystrophia, periosteal aplasia, fœtal rickets, achondroplasia, and osteopsathyrosis. Until their pathology has been more fully investigated, they may all be included under the one name of *osteogenesis imperfecta*. In some cases there has been found to be a defect in the endochondral ossification alone, in others in the periosteal alone; in yet other cases the anomaly consists, not in defect, but in excess of formation of one part of the bones, and hypertrophy of the cartilage with absence of true bone formation has been observed. The connection of these maladies with rickets is problematical; at the same time it must not be forgotten that true rickets is occasionally present at birth. The clinical manifestations of osteogenesis imperfecta consist in friability of the bones, showing itself in the occurrence of fractures, which sometimes are very numerous, and which specially affect the long bones; in stunting in the growth of the bones of the limbs, sometimes masked by excessive proliferation of the cartilage of the epiphyses; and in malformations of the cranial bones, such as the premature ossification and consequent shortening of the bones of the basis cranii (formation of the os tribasilare). The malady of the bones is not always incompatible with extra-uterine life, as is shown by the existence of adult achondroplasiacs. Cases have even occurred in which an achondroplasiac mother has given birth to an infant suffering from the same affection of the skeleton as the parent. Charrin and Gley have succeeded in experimentally producing lesions of the fœtal skeleton resembling rickets in rabbits by submitting the parent animals to the influence of the toxines of the bacilli of diphtheria, tubercle, and blue pus. In the human subject it must be confessed that sometimes the parents of infants born with fœtal bone disease have been apparently perfectly healthy. General fœtal dropsy and so-called fœtal rickets have been associated in the same case more than once.

Diseases of the Subcutaneous Tissue are not uncommon in the fœtus, and their characters are somewhat modified by the circumstance that the structures affected are in an incompletely developed state. The most important is *general fœtal dropsy or anasarca*. It is probably to be regarded rather as a symptom of various fœtal morbid states than as a distinct pathological entity. It is characterised by the existence of general anasarca, and by the presence of fluid in the peritoneal, pleural, and pericardial sacs; there is usually œdema of the placenta also. The dropsy may be so marked as to give a monstrous appearance to the fœtus and to obstruct delivery. In its advanced form the disease is quite incompatible with extra-uterine life. Family prevalence has been noted. The mother is not uncommonly dropsical also, and she may be the subject of cardiac, renal, or hepatic disease; hydramnios is an almost constant concomitant. It would seem that in some cases a purely fœtal cause of the dropsy is in existence, such as premature closure of the foramen ovale, absence of the thoracic duct, fœtal leukæmia, and cystic degeneration of the kidneys; but in other instances a maternal cause has to be looked for, such as anæmia, nephritis, or malaria. Some observers have regarded dropsy of the placenta as the starting-point of the whole morbid process. It is evident, however, that the dropsy is only a symptom, and may be due to a number of pathogenic circumstances. In its most deforming degree general dropsy is met with in the twin fœtus, especially in the allantoido-angiopagous twin or placental parasite. No treatment has yet been successful in preventing or in curing the malady, although chlorate of potash and iron in one case seemed to prevent the occurrence of premature labour, which is often the termination of a gestation in which the fœtus is dropsical.

Congenital cystic elephantiasis is a malady which in some particulars resembles general fœtal dropsy. In it, however, the subcutaneous tissue contains numerous cysts containing clear serous or curd-like matter, and the resulting deformity is greater. It may be generalised, but more commonly it is widespread without being quite universal in distribution; in other cases it is restricted to a special region of the body, such as the head and neck. It consists in dilatation, or in dilatation and occlusion of the lymphatic vessels and spaces; and the dropsy which results is generally gelatinous in its character—hence the name hydrops anasarca gelatinosa, sometimes given to it. Premature birth is the usual clinical result of this disease. The mother may show albuminuria and hydramnios, but the relation of these conditions and the fœtal state is not clear.

The name *congenital elephantiasis* is usually given, not to the cystic form which has just been described, but rather to the fibrous or telangiectatic or neuromatous varieties. In these there is a more or less localised thickening of the subcutaneous tissue, with nævus-like vascularity of the part, and sometimes with a distribution specially following certain nerves. The malady is quite compatible with extra-uterine life, and in some instances has been cured by elastic pressure. In some instances, as in the case reported by Mainzer (*Deutsche med. Wochnschr.* xxv. 436, 1899), the resemblance to elephantiasis Arabum of the adult is very close, and some have held that it is really the congenital form of that disease. A case has been reported in which a woman during pregnancy suffered on several occasions from lymphangitis; her infant at birth showed enlargement of one leg, and blood taken from the leg revealed groups and chains of the streptococci of Fehleisen; the conclusion drawn was that during the maternal lymphangitic attacks streptococci passed through the placenta into the fœtal circulation. This malady is associated in some way with the condition of unilateral hypertrophy which is occasionally met with, and in which the distribution of the enlargement is exactly limited by the middle line of the body.

Atrophic conditions of the *subcutaneous tissue* are sometimes found as congenital states. There may be a total absence of the subcutaneous fat, and this anomaly may persist until adult life is reached, when the individual affected is described as a "living skeleton." A condition which is probably closely allied is *cutis laxa* or *dermatolysis* (without tumour formations). The so-called "elastic-skinned men" exhibited at fairs and shows are instances of this curious condition of the subcutaneous tissue which microscopic examination has shown to be atrophy of the connective tissue bundles of the skin with a transformation of the firm tissue of the dermis into an unformed myxoma-like structure.

Fœtal ascites and peritonitis often co-exist in the fœtus; in fact ascites is rarely due to obstruction of the portal circulation in antenatal life, for the reason that the portal circulation is rudimentary then, and it thus happens that it is most commonly caused by peritonitis. Sometimes the abdominal distension thus produced is so great as to impede and even to prevent the birth of the infant altogether; as much as five or six litres of fluid have been found in the abdominal cavity. Usually the child is still-born or has to be killed during labour to diminish its bulk, but there are cases on record in which puncture of the abdomen has been followed by recovery. Hydramnios is not uncommonly associated with peritonitic ascites, and the association with malformations of the intestines and genito-urinary organs is also noteworthy. Fœtal peritonitis is not always accompanied with effusion, and a case has been reported in which there were traces of at least two attacks of dry peritonitis in the same fœtus. The cause of the peritoneal inflammation is seldom clear; in some cases it has been ascribed to syphilis, but in others there is absence of all signs pointing to this cause. The writer has met with an instance in which the mother had been subject to the infection of measles during the last weeks of her pregnancy, and the fœtus had peritonitis. Distension of the urinary bladder is sometimes associated with peritonitis; there may then be either hydramnios or oligo-hydramnios.

Fœtal endocarditis is of importance as the cause of morbus cœruleus in infancy and childhood. It must, however, be remembered that it is not certain that all cardiac anomalies are due to inflammation affecting the heart; some malformations arise directly as such in the embryonic period of antenatal life. The origin of fœtal endocarditis has been a subject about which doubt has existed; but it has recently been shown that it may originate from the passage of germs from the mother to the fœtus through the placenta. In a case of maternal erysipelas reported by Bidone, the fœtus was found to be the subject of streptococcic endocarditis, and it is probable that the streptococci found their way directly to the heart through the umbilical vein and ductus venosus. In the same way, in a case of fœtal tuberculosis it was evident that bacilli had made their entrance into the heart and had set up tubercular endocarditis.

The Transmitted Diseases of the Fœtus form an increasingly large group, and some maladies which were formerly regarded as idiopathic are now coming to be regarded as transmitted. Examples of this transference are found in endocarditis and elephantiasis, which have just been described. In addition to these

there are syphilis, the exanthemata, malaria, sepsis, tubercle, and possibly rheumatism. In an earlier part of this article the question of the mode of transmission of diseases from mother to fœtus was considered, and reference was also made to some of the peculiarities of the exanthemata as developed antenatally; these matters need not be returned to here.

Fœtal syphilis may be due to infection from a syphilitic mother through the placenta; apparently it may also be the result of sperm infection, without the intervention of the mother. A common result of fœtal syphilis is premature labour, and the pregnancy thus terminated may have been complicated with hydramnios. The infant may be dead at birth, and then shows the appearances of intra-uterine post-mortem maceration; or it may be born alive without any sign such as is usually regarded as syphilitic, but with congenital weakness, which usually proves fatal in a few days or weeks; or, again, it may be born with the bullæ of pemphigus, or the desquamation of ichthyosis, or with internal pathological changes, especially of the liver and bones, which are commonly regarded as syphilitic; or, yet again, it may show at birth malformations of various kinds, and may be even the subject of a monstrous development, circumstances which are now coming to be looked upon as the stigmata of embryonic syphilis. The placenta is often diseased, but it is difficult to be certain whether a typical syphilitic placental lesion exists; the afterbirth is often large, friable, anæmic, and shows on section fibrous patches, along with areas which have a fatty appearance. The umbilical cord is often thicker than normal. The protean character of the phenomena of adult syphilis is amply foreshadowed in fœtal syphilis. The treatment of fœtal syphilis may be commenced before birth, for if the mother or the father be syphilitic it has been shown to be advisable to administer mercury to the mother during the whole term of her pregnancy; even if the mother have apparently escaped the infection the same treatment is to be pursued for the sake of the fœtus, which may be syphilitic in virtue of infection conveyed direct to it through the spermatozoa of the syphilitic father.

Various *infectious fevers* may affect the fœtus *in utero*, and cases are on record of fœtal smallpox, measles, scarlet fever, varicella, typhoid fever, erysipelas, and influenza. In most of the reported cases the mother has been also a sufferer from the fever in question, but it does not appear to be necessary that she must always be so, for a mother rendered immune to smallpox by a previous attack or by vaccination may escape infection, while her unborn infant takes the malady and is expelled in time with the eruption or the cicatrices of variola upon him. The infection is probably always conveyed through the placenta, and in such a fever as typhoid, where the micro-organism is known and can be recognised, bacterial investigations make plain the avenue of invasion of the bacilli. It is true that fœtal typhoid differs in certain details, such as the rarity of the intestinal lesions typical of the adult form of the malady, but this is explicable on the general principles which govern intra-uterine infection. It has been recently shown that the blood serum of the fœtus of a woman suffering from typhoid gives the Widal typhoid reaction quite distinctly. There is some evidence to prove that infectious fevers in the fœtus may have effects which project themselves beyond the birth of the infant far into post-natal life; it is possible, for instance, that some of the so-called obstetric paralyses may have such an origin. This supposition is supported by an observation made by Osler, in which a pregnant woman suffering from typhoid fever died without the supervention of labour pains, and in the fœtus, removed from her uterus post-mortem, a well-marked hæmorrhage was discovered in the centrum ovale of the cerebrum. The blood effusion may have been due to the maternal typhoid; at the same time it may also have been syphilitic, for the woman was the subject of lues; but it certainly was not caused by compression of the fœtal head, for there had not been even the commencement of labour at the time the woman died. An interesting fact has been brought out with regard to fœtal measles, and probably the same is true of other fœtal fevers, to wit, that the infection of the fœtus and the mother would appear to be simultaneous, for the lesions in the fœtus, when it is expelled from the uterus, show the same stage of the disease as those in the mother; further, when intra-uterine death has occurred it would seem that at the moment of death the fever in the fœtus had reached the same stage as in the mother. The number of observed cases of fœtal fevers is as yet small, a circumstance due in no small degree to the difficulty of diagnosis, for it must be remembered that the physiological erythema and desquamation of the cuticle of the skin of the new-born infant are apt to prevent the recognition of fevers having these very cutaneous alterations as their leading features. At the same time, a very considerable literature has grown up around fœtal smallpox; in fact, it would seem as if all the possibilities with regard to this malady had been recorded, even to the extent that twins have been born, one of which was the subject of variola, while the other was apparently quite free from the infection. It may be also that, as in syphilis, so in the exanthemata, the incidence of the fever in the mother may determine in the fœtus, not the fever itself, but general changes in nutrition leading to stunting in growth, or even to actual malformations; possibly, also, as has been shown

in maternal erysipelas, the fœtus may suffer, not from the cutaneous manifestations of erysipelas, but from septic poisoning. In relation to fœtal sepsis it must not be forgotten that sometimes the fœtus has become infected from the vaginal secretions of the mother, either before labour, through premature rupture of the membranes; or intra-natally, during the progress of the body through the birth-canal.

Fœtal malaria is an occurrence the possibility of which may now be looked upon as proven. Women suffering from intermittent fever have given birth to infants with hypertrophy of the spleen, and showing in the blood the hæmatozoon of malaria; further, during gestation it has been recognised that the incidence of a maternal ague fit has been accompanied by active fœtal movements which could only be explained on the supposition that the unborn infant also was the subject of a rigor. Felkin has reported a curious case in which a woman, apparently quite free from malaria, gave birth to an infant with malaria, and showing the ague splenic enlargement; the father was seriously ill with malaria, and the supposition was that the fœtus had been directly infected by the semen. In cases of malaria in pregnancy the fear of causing premature labour or abortion by the administration of quinine has been probably much exaggerated; it is well to give the drug, not only for the sake of the mother, but also on behalf of the unborn infant.

Fœtal tuberculosis is a malady the existence of which has been doubted; but of late years a number of cases have been reported in which the conditions found were such as to leave no longer any reasonable doubt about the occasional occurrence of truly tubercular lesions in utero. In some of the cases actual tubercular lesions were discovered in the fœtus; in others, although there were no gross lesions, yet the tubercle bacillus was detected in the fœtal organs, and inoculations of animals with pieces of the fœtal organs and with blood from the umbilical vein set up in these animals undoubtedly tubercular processes. An excellent instance of fœtal tuberculosis, with lesions, was recently reported by Auché and Chambrelent (*Arch. de méd. expér. et d'anat. path.* xi. 521, 1899). It was that of a prematurely born, but living female infant, the product of a tubercular woman who died three days after her confinement. At the autopsy it was found that she had been the subject of advanced phthisis and of tubercular disease of the liver, spleen, intestines, mesenteric glands, and kidneys, but the genitals, organs, and peritoneum were free from infection. The infant, born about the sixth month of intra-uterine life, lived for twenty-six days in the couveuse, and then died, having lost weight, but having shown no other sign of illness. At the autopsy no peritonitis was found, and the intestine was free from lesions; but in the liver were numerous yellow granulations, in the spleen were crowds of the same punctiform granulations, and in the lungs were small numbers of grey, transparent granulations. The bronchial glands also were tubercular. Microscopic examination revealed an excessive number of tubercles in the liver, some caseated in the centre, with an enormous quantity of the bacilli of Koch. The spleen showed similar changes, there was tubercular endocarditis in the right ventricle of the heart. There were no giant cells. The placenta was also tubercular, and animals inoculated with pieces of it, and with the fœtal organs, developed tuberculosis. In this case there could be no doubt that the fœtus had become infected with tuberculosis. The reason why such cases are rare is probably to be found in the fact that in order to reach the fœtus the bacilli must be in the blood stream, and that they seldom are there unless the tuberculosis of the mother has become generalised. Further, many cases of fœtal tuberculosis may have been passed over because the lesions were looked for in the lungs, where, on account of the mechanism of fœtal infection, they are very seldom to be found. That the fœtal tissues are a suitable soil for the growth of the tubercle bacillus is proved by the extraordinary proportions of the bacillary invasion in the cases that have been reported. That the tubercle bacillus may be carried directly into the organism by the sperm has been affirmed, but manifestly the complete proof is surrounded with almost insurmountable difficulties.

The fœtus is liable to *toxicological states* transmitted to it from the mother through the placenta; but regarding these comparatively little is known. Much yet remains to be discovered regarding the effect of alcohol, absinthe, lead, mercury, copper, and the like taken into the maternal system upon the health of the unborn infant. It has been experimentally demonstrated that in the case of guinea-pigs the placenta has a tendency to store up copper and mercury, but not lead, which passes easily and completely through to the fœtuses; arsenic passes with greater difficulty, mercury apparently not at all, and copper, although it is accumulated in the placenta, also passes through it. It is interesting to know that lead, after it passes, tends to be stored up in the nervous system of the fœtus, while arsenic goes to the skin. From what has been experimentally determined regarding the teratogenic powers of alcohols injected into the hen's egg, it may be hazarded that it is possible that they may have a similar effect upon the mammalian embryo.

The *so-called* Traumatic States of the Fœtus include congenital fractures, dislocations, and amputations. The *fractures* that are met with in the infant at birth are usually due to the birth-traumatism, although they may have been predisposed to by truly antenatal fragility of

the skeleton, as in the various conditions to which the name "fœtal rickets" has been loosely applied; that true fractures may occur in intra-uterine life long before the onset of labour has been asserted, chiefly on the strength of the observation of the so-called cases of cured fracture noted after birth. That maternal falls and blows may by direct pressure or by contre-coup cause fœtal fractures, which afterwards consolidate in a vicious position and with much callus, is freely stated, but exact proof is not forthcoming. There is indeed reason to believe that some of these badly united fœtal fractures may be due not to external violence, but rather to the effect of amniotic adhesions or pressure. This, at any rate, would account for the association of the fractures with such malformations as the absence of one of the neighbouring limb bones, an association which is far from rare. Congenital *dislocations* may also be due to the traumatism of labour, but truly fœtal dislocations are in all probability rather to be regarded as the results of malformation than of injury. Dislocation of the hip would appear to be the commonest and the most important of the congenital luxations. According to one theory of origin, it is due to amniotic pressure upon the neighbourhood of the hip; according to another, there is an intra-uterine destruction of the tissues of the joint; another hypothesis regards it as caused by primary alterations of the nervous system producing either retraction or paralysis of the periarticular muscles; while yet another view is that it is really an arrested development of the acetabulum and surrounding parts, and not a dislocation in the true sense of the word. A great deal has been done of late years to correct this dislocation after birth, but as yet it is not settled whether the operative measures of Hoffa and Lorenz (enlargement of the acetabular cavity) or the bloodless method of Paci (reduction under chloroform and fixation by a plaster of Paris bandage) offer the better chance of success. Congenital dislocation of the knee, with or without absence of the patella, is not so common as dislocation of the hip; in it the head of the tibia may be displaced forward on the femur and give rise to hyperextension of the joint (genu recurvatum); occasionally, but rarely, lateral dislocation has been met with, and recently two cases have been recorded in which there was spontaneous and recurrent lateral luxation of the knee. Congenital dislocations of the patella, shoulder, and elbow have been noted, but they are very rare.

Congenital amputations are scarcely amputations in the usual sense of the term. The name is applied to the cases in which one or more digits, or even a hand or foot, or part of a limb is wanting at birth; and the explanation which has been commonly employed to account for the anomaly is that the umbilical cord or an amniotic band has become twisted round the part and has caused first a groove, then a deep furrow, and finally has entirely separated the portion of the limb lying distal to the constricting band. It is possible, however, that simple pressure of a tight amnion on the limb-bud may have hindered development, and so prevented the formation of the part. If this latter view be correct, the amputation is rather a malformation. It may be added that the condition has also been ascribed to a pathological state of the skin and subcutaneous tissue of a sclerodermic kind, and the disease called ainhum has been referred to as the corresponding morbid state in the adult. There seems to be little doubt that amniotic bands have something to do with the so-called spontaneous amputation; at any rate, amniotic adhesions and bands coexist with congenital amputations. It is a striking fact that in some of the recorded cases of amputation of digits there seems to have been an attempt at re-formation of the missing parts; for instance, at the end of the forearm there have been seen small fleshy tubercles supposed to be rudimentary fingers. In conclusion, it may be said to be doubtful whether congenital amputations belong to the pathology of the fœtus or of the embryo, whether they are pathological or teratological.

Intra-Uterine Death of the Fœtus,

Like post-natal death of the child or adult, may be due to very many *causes*. Attempts have been made, not always very successfully, to group these causes into maternal, paternal, and fœtal; probably in the great majority of cases the immediate cause of death is some lesion of the placenta, although it must be admitted that occasionally the placenta is apparently quite normal, even where there is good reason to believe that fœtal death had occurred some time before birth. Among the maternal causes of fœtal death may be enumerated endometritis and displacements of the uterus; acute febrile diseases such as pneumonia; acute infectious fevers, such as variola, cholera, typhoid fever, typhus; heart disease, nephritis; injuries, accidents, and operative and criminal interference; and, very specially, syphilis. A high maternal temperature, if it has been gradually reached, does not necessarily prove fatal to the unborn infant; but if the rise has been rapid as well as great, intra-uterine death usually follows. Purely paternal causes are little understood, although there can be no doubt about their existence; the best known is syphilis (the mother remaining unaffected); and others which have been enumerated are albuminuria, chronic lead poisoning, and old age. There seems to be no doubt that the various fœtal diseases that have been referred to above may all be the causes of fœtal death; but it would also seem that the fœtus may be very seriously morbid, and yet live on in utero

so long as the placental tissue is normal and the circulation in the umbilical cord not interfered with. In fact, as has been already stated, the integrity of the placenta is the great safeguard of fœtal life; consequently, when the placenta is the seat of hæmorrhages or of fibrous or myxomatous changes, intra-uterine death is very probable. Convolutions and knots of the umbilical cord, unless drawn very tight, do not produce fœtal death. Habitual death of the unborn infant is an occasional and a very distressing occurrence; its causes, in order of frequency, are syphilis, maternal anæmia, uterine diseases and displacements, alcoholism, renal and cardiac maladies, and "individual predisposition." Occasionally habitual alternate death of the fœtus may be recorded, and sometimes it has been noted that only fœtuses of the male or of the female sex died in utero, those of the other sex being born alive.

There seems now to be no doubt that intra-uterine death, like post-natal death, is followed by rigor mortis; but it is quite likely, that on account of the feeble development of the muscular system, the rigidity may be less marked and of shorter duration than that seen in the adult or child. If fœtal death occur between the third and fifth months, the commonest post-mortem change would appear to be mummification, a drying up and shrivelling of the fœtal tissues without putrefactive breaking down. If such a mummifying fœtus be subjected to pressure, either from a tumour in the uterus or (more commonly) from a living and growing twin, then it becomes flattened and is known as the *fœtus compressus s. papyraceus.* Maceration is the commonest result of intra-uterine death in the later months of gestation; it also is a non-putrefactive process. It is characterised by a softening of all the tissues and an infiltration of them with blood-stained serum; by desquamation of the cuticle, and by the formation of blebs or bullæ on the skin which contain yellowish fluid; and by the presence of blood-stained serum in all the body cavities. There may also be the changes due to the disease from which the fœtus succumbed, *e.g.*, in syphilis, the yellow line at the junction of the shaft and epiphysis of the long bones due to osteo-chondritis. The umbilical cord is often swollen and the placenta atrophic or œdematous, while the liquor amnii is of a dirty green colour from expelled meconium. Rarely true putrefaction occurs in utero with the development of gases; it is usually subsequent to rupture of the membranes, for the germs of putrefaction very uncommonly gain entrance save through the vagina of the mother. Calcification is very rare.

The *diagnosis of intra-uterine death* is one of the most difficult tasks that fall to the lot of the obstetrician. As a rule, the dead fœtus is expelled from the uterus about fourteen days after it has succumbed; then, of course, the expulsion of the dead and macerated body enables the diagnosis to be made. But sometimes the dead infant is retained for weeks and even for months in utero, and it becomes as important as it is difficult to be sure that death has occurred, and that we are not dealing with some other condition, *e.g.* a tumour of the uterus. There is no one certain sign of fœtal death similar to the certain sign of fœtal life (the hearing of the fœtal heart); and the diagnosis must be largely based on negative signs and symptoms. Among the circumstances which help in the formation of a diagnosis may be enumerated the history of the occurrence of fœtal death in a previous pregnancy in the same patient; the presence of syphilis or of some acute febrile or infectious disease, or the incidence of a traumatism, and, in a word, the existence of any well-known cause of fœtal death; the presence of various vague maternal sensations, such as a feeling of coldness and weight in the pelvis, absence of fœtal movements, especially following upon a period of unduly active movements; diminution in the size of the abdomen and uterus, retrogressive changes in the mammary glands and abdomen, occurrence of passing labour pains, impossibility of hearing the fœtal heart and of detecting fœtal movements; the disappearance of various grave symptoms, such as vomiting and albuminuria, which had been in evidence before the death of the fœtus; and, finally, it is possible that the occurrence of acetonuria and peptonuria may assist in the making of a provisional diagnosis. The treatment of intra-uterine death is intimately associated with the treatment of the causes of it (maternal, paternal, and fœtal). As a matter of experience, the birth of a dead fœtus does not seem to be attended with much danger, a fact which is explicable by the aseptic nature of the post-mortem change and by the involuntary changes in the placenta and uterine walls which usually precede the expulsion of the infant.

Affections of the Generative Organs

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Cancer, Fibroid Tumours, Ovarian Tumours in Pregnancy

The course of pregnancy and of labour may be seriously affected by anomalous conditions of the uterus and vagina due to defects of development, to abnormal positions of the organs, and

to pathological changes in their tissues. When such cases occur, questions of grave importance may arise as to the risks which the patients run, and the proper means of management. What concerns us at present is not so much the discussion of the ever changing views regarding treatment, as the clinical recognition of those defects, and the experience which has been accumulated as to their influence on pregnancy and dangers that may occur, or in other words, the knowledge upon which the solution of questions in individual cases should be based.

1. Malformations of the Uterus and Vagina.—The nature of the various anomalies of the generative tract and their clinical features can be readily understood by remembering the origin and development of the organs from the two Müllerian ducts. All the varieties are explained by the retention in whole or in part of this duplex character, and by an imperfect development of one half in comparison with that of the other. Normally the union is complete from below as high as the Fallopian tubes, between which is developed the fundus uteri, whilst by total absorption of the septum the uterine body, the cervix, and the vagina form a single canal. In any of these respects, throughout, or at any one part, the development may be imperfect, and so are produced the various anomalies that have now to be noted.

The *fundus* alone may be imperfectly formed, and show a flattening or depression instead of the usual convexity—*uterus arcuatus*, *s. corduatus*. This defect may be associated internally with some remains of the septum. Pregnancy is not affected thereby, but it may have an influence in the production of malpositions of the head (Auvard), and where a partial septum is present lead to difficulty with the placenta.

Other abnormalities arrange themselves under various types, the distinctive characters of which may be more or less pronounced in different cases.

Uterus Septus.—Here the external formation is normal, with perhaps a slight increase in width, but the septum remains, dividing the cavity more or less completely into two. The septum may be only partial, *uterus subseptus*, or it may extend to the internal os, or even into the cervical canal, but the external os is single.

Uterus Bicornis.—Here the duplex character is indicated externally by the uterine body being divided into two distinct cornua, at a varying height above the cervix, each horn terminating in its own Fallopian tube, but with a common cervix, the canal of which may be single or double.

Uterus Didelphys.—In this rarest variety the separation is complete, two single uteri lying side by side, each possessing its own os uteri and cervical canal, but only one tube, ovary, and round ligament. The vagina may be single, or duplicated in whole or in part. It is doubtful if pregnancy occurs in this variety.

Uterus Unicornis.—Where one half has been well developed, presenting a spindle formation ending in the Fallopian tube, the other half remaining in a rudimentary state or imperfectly developed form.

In any of the above duplex conditions one half may have no communication with the general canal. The menstrual fluid in that half will then be retained, giving rise to a tumour the nature of which may be difficult to diagnose.

The *vagina* may be double throughout, even when the cervix and uterus are single; or partial remains of the septum may be present, forming a band of tissue across the canal.

Such anomalies in development do not interfere with the possibility of pregnancy so long as ovulation takes place, and there is no occlusion in the generative tract. If, on the other hand, the impregnated ovum is prevented entering the uterine cavity, the case belongs to ectopic gestation. In the same category must be placed the rare cases where the ovum has been lodged in an imperfectly developed half, having no communication with the fully developed horn or the vagina. In such a case rupture into the peritoneal cavity is likely to occur about the third or fourth month, or if the fœtus dies before rupture, it may be indefinitely retained.

Diagnosis.—With a double or one-horned uterus, pregnancy is not uncommon—much more frequent, indeed, than is recognised. From various circumstances, the condition may very readily be overlooked. As a rule, the course of pregnancy is not disturbed, and labour is easy and natural, and in some forms, as in the uterus septus, the abnormality can be discovered only when occasion calls for the introduction of the hand into the uterus. Even where it might be possible in the non-gravid state, or early pregnancy, to recognise the condition from the abnormal shape of the uterus, this may be considerably obscured in advanced pregnancy or in labour. In a uterus unicornis the spindle shape is much altered, and in uterus bicornis, where one horn only is pregnant, it may lie in front of and conceal the other.

A double vagina calls for careful examination of the uterus; for although it may be present with normal conditions above, it is always a possible indication of other anomalies. The presence of two os uteri and complete double vagina are sure signs of a duplex uterine cavity.

When both divisions are pregnant, a diagnosis

can more readily be made. A furrow more or less marked may be detected between the two halves where they are united in one body; where the horns are distinct they diverge from one another, more especially when the uterus contracts.

Other conditions suggestive of a double uterus are referred to below.

Phenomena and Course of the Pregnancy.—The pregnancy may be single or double, spoken of as unilateral or bilateral, *i.e.* where one or both horns are occupied. Twins have also been met with occupying the same horn, and in the uterus unicornis. When both cornua are occupied, the ova may have been impregnated at different times, even at considerable intervals (*superfœtation*), and the fœtuses may show at birth a corresponding difference in development, or the one fœtus be retained *in utero* for some weeks after the birth of the first. In all such remarkable cases, a careful examination of the uterus should be made to ascertain the presence or not of a duplex uterus.

When one half of a double uterus only is gravid, the other, to some extent, shares in the hypertrophy of the walls, and a decidua may be formed in it. Under such circumstances it is possible that considerable or repeated hæmorrhage may occur during the pregnancy, from the empty half, without endangering the gestation. An explanation may thus be afforded of cases where it has been stated that menstruation has regularly recurred during pregnancy, or that the woman menstruated only while pregnant.

During labour, both divisions have been felt to contract and relax simultaneously. Where there were two os uteri, that of the empty half has been observed to dilate and a decidua to be expelled. At the same time each horn may be functionally distinct and contract independently of the other.

Though the labour is generally easy and natural, yet anomalies are more frequently met with. That a greater tendency to abortion exists does not rest on satisfactory evidence. It is in labour that difficulties are likely to be experienced. The increased inclination of the uterus is liable to affect the position of the head; breech and transverse presentations are more frequent than in single uteri. The labour may be rendered tardy from the direction of the uterine force not closely corresponding with the axis of the brim. The upper portion of the horn differs from the natural fundus uteri; the walls may be thinner, and the risk of rupture is greater.

Difficulties with the placenta are liable to occur when it is situated in the upper part of the horn, or is attached to the division in the uterus septus. Hæmorrhage in the latter case is likely to follow the separation. When the septum is incomplete, the placenta has been found in the one division, whilst the fœtus in its development has passed into the other.

Bands of tissue in the vagina have caused difficulty in the second stage of labour. Where such is the case, it is better to divide them artificially than leave them to be torn by the natural forces, the risk of the laceration extending into the vaginal wall being considerable.

2. Abnormalities in Position and Relations of the Gravid Uterus.—The gravid uterus has, at rare times, been found in strange quarters, as in the sac of *an inguinal or femoral hernia*. Under such circumstances gestation is likely to be arrested; cases have, nevertheless, been recorded where a child has been delivered by hysterectomy and survived. Considering the risks to the mother, such cases, as soon as recognised, would in the present day come at once under the surgeon's knife.

In the later months of pregnancy the fundus uteri may come into relation with a pre-existing *umbilical hernia* and partially protrude into the sac; in one case two-thirds of the organ is said to have so passed. Labour will not be complicated unless adhesions have been formed interfering with retraction; in one recorded case retention of the placenta in the hernial sac caused difficulty.

A uterus liable to *prolapsus* or *procidentia* may become pregnant. With ordinary care the displacement disappears with the increasing size and elevation of the organ. A complication may arise where the enlarged uterus by a sudden strain has been forced out, and, from its size, difficulty is experienced in returning it. If unreduced, symptoms of incarceration and abortion would ensue. In older writings, cases are recorded where pregnancy in a supposed prolapsed uterus had advanced to full term; but these have evidently been cases where the prolapse has been limited to the cervix alone, from great hypertrophy, the uterine body remaining within the pelvis. The position of the fundus in the cases recorded is not stated. Acute œdema of the cervix may also simulate prolapsus during pregnancy, and also after labour. Prolapse of the anterior vaginal wall may be a source of much discomfort during gestation, and demand attention in the management of the labour.

A uterus that has been the subject of complete prolapse, but now retained in the pelvis by pregnancy, is liable to assume a retroverted position and cause further trouble. This must be borne in mind when replacing a prolapsed gravid uterus.

Versions and Flexions.—At one time there was a tendency to impute prejudicial effects in pregnancy to *flexions* of the uterus, and so regard as pathological a condition that often exists, but in no way interferes with gestation. The same may be said of *anteversions* in the earlier months. Frequency of micturition and

other inconveniences may arise from the pressure of the enlarging uterus, but these diminish when the organ rises above the brim; incarceration in this position does not occur. Some degree of flexion is almost always present in cases of version, but it is the latter only that endangers the pregnancy, as is shown by the relief obtained by correcting the version, whilst the flexion is left, and in fact cannot be treated.

Pendulous Abdomen. — *Anteversion of the Uterus in the Later Months of Gestation.*—In the earlier months of gestation anteversion, as before stated, causes no difficulty; it is in the later months the condition becomes of importance, and is then more graphically termed and better known as pendulous abdomen. It is in general due to relaxation of the abdominal walls owing to previous pregnancy, and is certain to be present when separation of the recti muscles has occurred either from child-bearing or after cœliotomy. It may, however, be present in a primigravida, and is then suggestive of narrowing or other deformity of the pelvis.

The course of pregnancy is not disturbed thereby, but difficulties and even grave complications in labour are liable to arise from the resultant of the uterine forces deviating considerably from the line of the axis of the inlet. Tardy dilatation of the os uteri, malposition of the head, the presenting part refusing to enter the brim, consequent exhaustion of the uterus from ineffectual action, and even rupture of the uterus may occur.

Placing the patient on her back, elevating the uterus, and applying a binder, is the proper treatment.

Retroversion of Gravid Uterus.—Although retroversion may at times be the cause of sterility, it is no certain barrier to impregnation. With the natural growth and elevation of the uterus, the displacement is often undone, and the pregnancy is not interrupted. But this is not always the case; abortions are frequent and often repeated when the uterus is retroverted. This can readily be understood; for the mucosa and blood-vessels of such an organ are not likely to be healthy, or, if previously healthy, the uterine circulation is so impeded that hæmorrhage is very liable to occur.

The usual pelvic symptoms—discomfort, fulness, weight, dragging, or pain—gradually increase, relieved by lying down. Amenorrhœa and other signs of pregnancy are present, but obscured, it may be, by slight shows of blood at intervals. Bimanual examination reveals the readily recognisable signs of retroversion.

It is never safe to trust to the natural process of righting the position. In uncomplicated cases during the first three months the version can often be corrected *manually*, without difficulty, and a Hodge or ring pessary carefully fitted. Examination must be made from time to time to ascertain that the pessary has not got displaced. It is unnecessary to persist in attempts to get the uterus into the theoretical "normal" position: all that is necessary is to secure, if possible, that the fundus is on the same or a higher level than the cervix; the body may still be retroposed; if the tendency to prolapse be corrected by a pessary, nature will do the rest. The patient should lie as much as possible in the semiprone position, and avoid the necessity of straining at stool by the regular use of saline aperients.

Incarceration of the Gravid Uterus.—When a case of pregnant retroverted uterus has not received attention, and the natural growth and elevation has not corrected the version, more serious pressure symptoms arise, indicating *incarceration*. They begin in the third or early in the fourth month, when the increasing bulk of the uterus becomes too great for the pelvis. This is more likely to happen when a marked degree of prolapse has been associated with the version, or a projecting promontory narrows the inlet. The patient, who previously has complained of the frequency of micturition, may find herself unable to pass water. The complete retention may come suddenly, or after several successful efforts. The bladder becomes distended; there is acute hypogastric pain, paroxysmal in character. The nature of the case may be overlooked from the patient stating she has been constantly passing water—the dribbling of an overfilled bladder. General distension of the abdomen, with tenderness on pressure and vomiting, may further obscure the case.

When unrelieved, serious complications have occurred: cystitis, sloughing of the mucous membrane, and gangrene of the muscular coat of the bladder, distension of the ureters and kidneys, with consequent uræmia or septicæmia, have all been recorded.

The Diagnosis.—Given the history of a possible pregnancy in the third or fourth month, urinary troubles, a central abdominal tumour which the use of the catheter has demonstrated to have been a distended bladder; per vaginam, the pelvis occupied by a rounded spherical tumour which may vary in consistence, and the cervix high up in the front, lying against and looking towards or above the symphysis—the diagnosis should not be difficult.

Differential Diagnosis. — Nevertheless, the same effects may arise under different circumstances. A tumour similar in size, shape, and position, and causing retention of urine, may be formed by the sac of an extra-uterine pregnancy, or the hæmorrhage due to a tubal abortion, by a retropubic abscess, or by a small ovarian cyst distending the pouch of Douglas. In the first two cases there will be some evidence of pregnancy, and the differential diagnosis is mainly dependent upon careful bimanual examination. When the tumour is outside the body of the uterus the latter is pushed bodily

forwards, and may be defined in front of the tumour, and the axis of the cervix directed *downwards* instead of forwards towards the symphysis pubis. In the other conditions, besides the same physical signs, there is not likely to be amenorrhœa or other signs of pregnancy.

Treatment.—The first step is to empty the bladder. Difficulties are sometimes met with here. The points to attend to are, to use a gum elastic catheter, not a glass or other rigid one, or one of soft indiarubber; the passing the instrument must be carefully guided by a finger in the vagina passed well up in front to the cervix; and, lastly, to remember that from the drawing up, stretching, and elongation of the urethra, the instrument may have to pass a greater distance than usual before the water is reached.

It is not well in all cases to proceed at once to attempt rectification of the position of the uterus. It is advisable to have the bowels well acted upon by a saline purge, not merely emptied by enemata. Further, the pelvic vessels are certain to be distended, and the tissues more or less œdematous. These conditions are relieved by the saline, and by rest for some hours in the semiprone position. After this preliminary treatment, rectification should be tried.

With the patient in a favourable position—semiprone or knee-shoulder—and in some cases under an anæsthetic, the operation is at times readily effected; at others, repeated trials have to be made. After a failure of this kind, it not unfrequently has been found that the uterus has spontaneously rectified itself, or that the desired effect has been obtained by the introduction into the vagina of a hydrostatic bag or elastic ring pessary. No doubt the manipulation in the futile attempts may have modified the conditions that caused the incarceration, but the fact that means so simple have effected replacement, after careful taxis has failed, is interesting and instructive. The lessons it teaches are, that the difficulty in most cases is not to be overcome all at once by strenuous efforts; that the primary idea under which to work is not to secure immediate elevation of the fundus above the brim, but to *correct the version and prolapse*; effect this, even partially, and nature will likely do the rest. To accomplish the necessary rotation of the uterus upon itself with a minimum of force, two forces are required: one acting upwards on the body of the uterus, the other downwards on the cervix. The latter is the more difficult to apply. Through a thin abdominal wall pressure may be applied externally above the pubis, or the finger internally may exert sufficient pressure on the cervix, but it is difficult to get a purchase in this manner. Failing these, a hold may be taken of the anterior cervical lip with a button volsellum; it does not wound the surface, but no great force should be exerted, lest the tissues be torn. It is not the amount, but the direction of the force that is effectual.

Very instructive also are the good results obtained by Professor Playfair, who used a hydrostatic bag in the vagina, and by Professor Japp Sinclair, who introduces an ordinary watch-spring ring pessary. He reports fifteen consecutive cases without a failure. Now such instruments cannot exert a force sufficient of itself to elevate the fundus, such as is artificially applied in the ordinary taxis. An elastic pessary lying in the vagina may exert some pressure, but the direction is backwards towards the hollow of the sacrum, not upwards towards the brim. Then again, in seeking to explain their action, the fact must not be lost sight of, that, after a futile attempt at rectification, the natural processes have spontancously righted the uterus. It is the latter that are the active agents: the action of a pessary is passive. A pessary is not a lever, as has often been supposed; it may, however, act as a fulcrum. During pregnancy, the natural forces normally tend to elevate the fundus, even when retroverted; but in certain cases this tendency is reversed—the cervix is elevated, the fundus is more and more depressed. The factor in such cases that has been hitherto disregarded is in all probability the varying degrees of prolapse that are associated with the retroversion. By the introduction of artificial support from below, the tendency to movement in the wrong direction is corrected, and the natural powers so enforced rectify the malposition.

The disastrous effects that occasionally have been met with arise from carelessness in examination, and latc recognition of the real nature of the case. The more the above facts are recognised, the less strenuous will be the attempts at immediate rectification, and rarer the occurrence of irreducible cases.

Irreducible Retro-displacement. — With the present greater appreciation of the value of rest, the use of anæsthetics, and the greater knowledge of the natural powers, aided, it may be, to overcome the obstruction, it must indeed be rare to meet with a case that must be pronounced irreducible and demands further interference. Yet such cases have, and possibly may again occur. Strong adhesions may be the cause. Left unreduced, serious consequences are certain to arise.

Abortion may be induced; but the conditions render the operation difficult and liable to be incomplete, and to be followed by inflammation and sepsis. Olshausen and others have removed the whole uterus by the vaginal method. But in the present day, if the more rational methods have failed and urgent symptoms threaten, cœliotomy, separation of any adhesions, and

pulling up the uterus, is undoubtedly the preferable operation, and one that may not interrupt the pregnancy. This has been done eleven times by Jacobs, surely in some cases unnecessarily, and once by Murdoch Cameron of Glasgow, and others. But cases requiring such a proceeding must be very rare.

Sacculation of the Gravid Uterus.—*Partial Retroversion or Flexion.* — In the advanced months of pregnancy, cases arc at rare times met with where the cervix uteri is displaced upwards close behind or above the symphysis, difficult to reach, and the pelvis occupied by a tumour which careful examination proves to be the posterior uterine wall containing the presenting part of the fœtus, whilst above the uterine body and contents form a well-marked abdominal tumour.

The condition can be realised by supposing a retroverted pregnant uterus which has been but partially righted, the cervix high up behind the pubis, the lower part of the posterior wall remaining in its abnormal position, whilst the anterior wall has been developed and stretched sufficiently to allow the upper pole of the fœtal body to rise into the abdomen.

Such, too, is the probable origin, in some cases, of the condition. It has, however, been found associated with an ovarian tumour (Croom), and with fibroid tumours of the uterus (Dührssen and Munro Kerr). Scanzoni has observed it along with pendulous abdomen, to which condition he ascribes it.

Pressure symptoms are liable to be troublesome. Barnes reports a case where the ureters became involved.

Spontaneous rectification has been observed before and also during labour. The pelvic tumour has also been successfully pushed up, and the cervix brought to its normal position, during gestation. Labour may not ensue till full term, but is liable to be premature.

An analogous condition affecting the *anterior* wall may be met with. The cervix is then far back and drawn upwards—to be reached only by introduction of the hand into the vagina under anæsthesia. Such cases have been mistaken for pathological occlusion of the os, and the cervix incised.

If reduction has not been effected before labour sets in, the first stage is certain to be protracted. The uterine force cannot act directly on the os uteri. The presenting part should be pushed up, artificial dilatation of the os used, and the anterior lip drawn forward or the posterior pushed back, according to the condition. Version or the forceps may be employed, careful support being given to the cervix during extraction.

Pathological Changes

Cancer of the Uterus and Pregnancy.—During the child-bearing period, cancer of the *body* of the uterus is very rare: pregnancy in this condition probably never occurs. If it did, an early abortion would almost certainly result, and would likely be unrecognised, as the pain and hæmorrhage would be assigned to the pre-existing disease, and the evidence of the conception lost in the discharges.

The same, in all probability, often happens when the *cervix* alone is involved. But that pregnancy may occur under this condition and advance to the full term there is but too ample evidence.

To the medical attendant, a case of pregnancy complicated by carcinoma of the cervix uteri must be throughout a cause of anxiety, so numerous are the risks, and uncertain the course of gestation and progress of the disease. At the same time, the conditions and results in different cases are so varied, that it is difficult to arrive at any reliable conclusions to guide in determining what in a special case will be best for the patient. An analysis of a large number of cases is the only reliable aid. This will be found in a valuable paper by G. Ernest Herman in the *London Obstetrical Transactions*, vol. xx. p. 191, 1878. Since it was written, however, the advance of surgical technique and skill has modified some of the conclusions arrived at.

It is now generally accepted that pregnancy *stimulates the activity of the morbid growth*, though at one time the opposite was believed. Whether the pregnant state, apart from any injurious effect of labour, hastens or retards the general constitutional changes which directly lead to a fatal issue, has not been determined. Certainly the presumption is that the prognosis would be more favourable without the pregnancy, and not made worse by the gestation being ended. The improvement that has often been observed after the puerperium is past supports this view.

The frequency of *abortions* and of *still births* is increased, and labour is more apt to be *premature*; the prospects of the child are therefore considerably lessened. These considerations must have weight in considering, in a case, the advisability of artificially emptying the uterus.

In the early months of pregnancy, and in an early stage of the disease, where one otherwise would recommend *hysterectomy*, the existence of the pregnancy should be no barrier to the operation. Delay is dangerous, as by the time the child is viable, the disease, as has happened, may have so advanced as to contra-indicate any attempt at removal of the organ. But in the later months, if the child has nearly reached a viable age, and hysterectomy is deemed suitable, the mother should take the risk of a short delay rather than wholly destroy the chances of the child.

Where hysterectomy is inadvisable, or the consent of patient and friends is refused, it may

be possible to remove the greater part, if not all, of the diseased structure where it is still circumscribed and the neighbouring tissues are not infiltrated. The dangers are not greater than in the non-gravid condition, and the risk of inducing abortion has proved to be slight. The prospects of the patient are greatly improved; gestation may go on to full term, and end in a labour easier than otherwise it would have been. Even if abortion did follow, it is the step which, without the operation, might be advisable. It is now generally accepted that before the sixth month, where the disease cannot be wholly removed, the *induction of abortion* offers the best chance to the mother.

The tolerance of the pregnant uterus to operations on the cervix enables one to meet a complication, where it has been decided to allow gestation to run its course. Severe or repeated hæmorrhages may seriously affect the patient's strength. The ragged and cheesy surface can be removed with the curette, well into the canal, but not to interfere with the internal os and membranes, and the cavity thereafter plugged with iodoform gauze. The bleeding can in this way be checked, and the pain from which the patient has suffered is often relieved.

In thus operating on the cervix, even when removing considerable portions of the diseased structure, bleeding is seldom considerable, and is easily restrained by pressure of the tampon.

When the pregnancy has advanced beyond the sixth month, one of three courses can be adopted:—1. To induce premature labour; 2. To wait till labour ensues spontaneously, and then meet the difficulties as they arise; 3. Cæsarean section when labour begins, or previously at a determined time. The position of the latter operation is very different from what it was ten years ago. It can now be performed with comparative safety. It is no longer the last resort where it is impossible to deliver the patient by other means, but it is to be preferred in all cases where delivery by the natural passages, though possible, is certain to entail the dangers of a protracted labour with much bruising and tearing of the tissues. The interests of the child also are better safeguarded.

The question of treatment, therefore, turns on forecasting the probable difficulties of the labour, and the prospect of life to the mother should she survive the confinement.

When the *morbid growth is limited* and a fair portion of the cervical tissue is free, the anticipation of difficulty is not great. It has not infrequently been found that when even the whole circumference of the os uteri was involved, dilatation has been accomplished by the natural efforts, tardily it may be, but successfully, and the patient made a good recovery. It would appear that difficulty in full dilatation does not depend upon the extent or size of the growth so much as upon its consistence; if the cancer growth be soft, and the surrounding tissues but little involved, it will not offer much resistance to the uterine force, aided if need be by the forceps. The expansion in most cases is finally attained by *fissuring*, and this does not seem to increase the dangers to the mother.

Following this indication of nature, *incisions with scissors* have been made with marked benefit, and are probably to be preferred to trusting wholly to accident. This procedure has proved more reliable and safer than attempts to dilate with indiarubber bags.

There are cases, however, where the risks likely to be encountered are more hazardous than the *Cæsarean operation*, especially where the morbid growth is extensive and hard, or the neighbouring tissues are infiltrated. In no case should the decision be made to trust to natural efforts without a careful *examination per rectum*, as well as by the vagina; much more information can thus be gained of the amount and consistence of the parts involved.

It should be noted that in labour it has at times been possible to remove considerable portions of the cancerous growth without injurious effect, and labour in this way accomplished. Successful cases have been recorded, but also unsuccessful, and in these the feeling must be that in the present day Cæsarean section would have been preferable.

It has been argued that in cases where difficulties are likely to be met from the state of the cervix, *premature labour* should be induced, as the obstruction will increase rather than lessen by waiting, and the fœtal head will be larger. To the mother, any benefit to be gained is slight, whilst to the child, whose claims are now paramount, the risks would be increased by prematurity and the difficult delivery. Cæsarean section conserves its interest, and can be done if the dreaded increase in the obstruction has occurred, and is to be preferred where otherwise craniotomy is the only resource. In the milder cases, forceps are safer to mother and child than turning.

Fibroid Tumours and Pregnancy.—Small round myomata are often present in the pregnant uterus, and cause no trouble. Even when multiple, if distributed and not masked together, or if single and of moderate size, they may not influence the pregnancy or complicate labour, provided they do not distort the uterine cavity or obstruct the passages, and no untoward change occurs such as described below. These cases do not call for interference. Uterine action is not impaired, as has often been stated; but there is risk of malpresentations. Hæmorrhage, too, is likely should the placenta be attached in the situation of the tumour. The bleeding, in such a case, might be difficult to control; probably this is one condition where packing the uterine cavity would be preferable to the ordinary hæmostatic measures.

Different is the prospect where the tumour is situated in the lower uterine segment or cervix, and lying in the pelvis or immediately above the brim, dangerously narrowing the canal, or preventing the fœtal head engaging the brim. Cases have been recorded where a tumour so situated has in labour been drawn up by uterine action or pushed up by the attendant, and the labour successfully terminated. So fortunate an occurrence as the first cannot be reckoned on, whilst the use of strong artificial force has proved injurious or futile as often as it has been successful. If the tumour is in the anterior wall, it may possibly be drawn or pushed up, as this accords with the natural mechanism of labour; but in the posterior wall no such natural movement takes place, elevation is then possible only when the tumour is peduncular, and such a condition might be inferred when it is known from a recent vaginal examination that previously no tumour occupied that situation.

The presence of uterine fibroids is recognised as a cause of *repeated hæmorrhages* during gestation, and also of *abortion*. When the uterine cavity is distorted by the mass, or the cervix involved, much difficulty may be experienced in completely emptying the uterus. When curetting has been necessary, or there is fear that small portions of membrane have been retained, thorough washing out the uterus should be repeated daily till all risk is past.

Uterine myomata increase in size with the growth of the uterus, and when all goes well they share in the process of involution. But during this growth serious effects may ensue, causing much suffering, and even endangering life. Some are purely mechanical; others are due to pathological changes.

Pressure Symptoms.—Exhausting pain, obstruction of bowel or bladder, or local œdema, may set in. *The relation of a myoma to the lower segment or to the pelvis* may change, so that, whilst at an early period of pregnancy no obstruction was indicated because of the high position, yet before full term is reached it may have descended so as dangerously to narrow the canal.

Pathological changes in the myomata not infrequently occur. *The rapidity of the growth may be out of all proportion to that of the uterus.* The tumour takes on an independent action, leading to pathological changes in its structure—softening or cystic—and to morbid action in contiguous tissues, especially peritonitis with adhesions. The tumour may continue to grow after delivery, or degenerative or septic changes ensue, with consequent blood-poisoning, or extreme emaciation as in malignant affections.

Subserous pedunculated myomata are especially liable to degenerative changes, and to contract adhesions to neighbouring organs—probably from twisting of the pedicle or other interference with nutrition whilst under the stimulus of pregnancy. They are apt to slip down into the pelvis behind the uterus, or to one side, and form a formidable complication in labour. Their treatment will be discussed later.

Obstruction to the passage of the child, then, is not the only point in the determination of the question of interference. The liability of pathological changes in the tumours add fresh complications. Undue rapidity of growth, softening, or inequality of consistence, are important elements that of themselves may warrant operation; still more if the woman is exhausted by pain or sickness, febrile attacks, or emaciation.

Treatment.—The improvements in surgical technique, the increased knowledge gained by abdominal sections, and the greater attention paid to the subject in recent years, have considerably modified the opinions and management in vogue in previous times. In this paper the endeavour has been to give the basis upon which the question of interference is based; the time and mode of operation must vary from time to time as experience increases. Various points as to treatment have been referred to above; the following are in accordance with the results of the operations recorded during the last twenty years. Details of the operations cannot be discussed.

As indicated above, there are cases that may be allowed to advance to full term. In these unexpected difficulties may arise in the course of labour. It should be borne in mind that a protracted labour, complicated by myoma ended with difficulty by version, forceps, or embryulcia, is as dangerous, if not more so, than Cæsarean section, or Porro-Cæsarean operation performed under favourable circumstances—that is, before the patient's strength has been exhausted, or futile or protracted attempts at delivery have been made.

"On reviewing Lefour's cases, we find that of 66 which terminated unassisted, 32 ended fatally, or a mortality of 48·4 per cent. Of 35 cases of versions, 21 died, or 60 per cent; while the percentage mortality attached to forceps and embryotomy is 25·9 and 55·5 respectively." Dr. John Philips, *B. M. Journal*, June 21, 1888.

Induction of abortion or of premature labour naturally suggests itself as the least formidable operation. Experience has shown that it is not so, especially where the cavity is distorted. The placenta is liable to be retained, and in some cases it has been impossible to reach it by the hand. The tumour may also make the use of ovum forceps or the curette extremely difficult. The risk of septicæmia is great. It is not an operation to be adopted without careful consideration. Moreover, a subsequent operation will in all likelihood be necessary.

Removal of the Tumours during Pregnancy.—The advance of surgery has now provided the

means of averting the dangers that are imminent and allowing the pregnancy to advance to term. According to their relation to the uterine walls, tumours have now been successfully removed by several skilled operators, and in a large proportion of these without interrupting the course of gestation. Peduncular myoma that are growing rapidly, or undergoing change in structure, or causing pain, should always be removed. The pedicle is liable to become twisted. *Myotomy* in the subserous form has in a few cases been done, but it involves much greater risks.

When the local or constitutional disturbances are severe and threaten to be serious, by *cœliotomy* the condition can be exactly determined. Cases have occurred where a peduncular or otherwise accessible tumour has been found and removed, the urgent symptoms relieved, and gestation not interrupted. In other cases, it proved necessary to remove the uterus with the contained fœtus intact, "Müller's ablation." When uterine conditions preclude pregnancy advancing successfully, this operation should be performed early. It has afforded better results in skilled hands than other operations in the later months.

When the fœtus has reached a viable age, induction of labour, with a possible difficult delivery, has not given good results. No advantage is thus to be gained. When there is room to hope for a favourable delivery, natural labour may be waited for. If there is a fair probability of success with *forceps* they may be cautiously used, but not long persisted in. The case is different from ordinary narrowed brim. The pressure on the resisting structures has caused subsequent sloughing and disintegration with septicæmia. The question of attempting to push an obstructing tumour out of the way has already been discussed. *Tumours accessible per vaginam have been removed during labour.* Such cases must be rare. In judging of the advisability of making the attempt, it must be remembered that, though successful, and the labour terminated, the uterus is left in a condition where metritis and peritonitis are most liable to occur. Peduncular tumours from the interior of the uterus, when found, should be removed.

Cæsarean section, or *Porro's operation*, at times comes to be the only resource to save the woman's life. The mortality in the past has been great; but this has been largely due to the fact that, in the majority of cases, the operation has been done only after the patient has been brought into a very unfavourable state by delay and other unsuccessful attempts at delivery. If performed only as a *selective* instead of *compulsory* operation, the chances are greatly improved. Simple Cæsarean section, under the circumstances, involves greater risks: from the uterus being left, a subsequent operation might become necessary, and there is a possibility that the woman might again become pregnant. The Porro-Cæsarean is in every way preferable. The only condition where it would be impossible is where the cervix is so involved in the morbid growth that a stump cannot be formed. The Cæsarean section alone must then suffice. (See also vol. v. p. 304.)

Ovarian Tumours in Pregnancy. — An ovarian tumour constitutes a formidable and, as seen from the large number of cases recently published, a not uncommon complication of pregnancy. Any of the forms of ovarian tumour may be met with, but the relative frequency of the various kinds is somewhat different in pregnancy. The proportion of dermoid tumours, for example, is much greater than in the non-pregnant. According to Olshausen, they constitute about $3\frac{1}{2}$ per cent of all cases, while in a series of 700 cases associated with pregnancy the tumour proved to be a simple or multilocular cyst in 477, a dermoid in 165, sarcomatous or carcinomatous in 40, and in 18 a fibroma or solid adenoma. Various reasons have been assigned for the disproportionate number of dermoid tumours; but whatever the explanation, the fact is important, as they form a more serious complication of pregnancy than the ordinary cystic forms, owing to their greater liability to cause inflammatory symptoms.

When associated, pregnancy and ovarian tumour react unfavourably on each other, and their reciprocal effects are of clinical importance. The presence of the tumour undoubtedly predisposes to premature interruption of the pregnancy, but this tendency has been much exaggerated. At any rate, it is exceptional for abortion to occur if the tumour itself escapes complications. In several cases, incarceration of the uterus has been caused by an ovarian tumour; while in the end of pregnancy the tumour, more particularly when large, may prevent the head from entering the pelvic brim. Even the largest tumours do not seem materially to interfere with the development of the fœtus. On the other hand, pregnancy predisposes in a marked degree to the various accidents to which ovarian tumours are liable. For example, tortion of the pedicle cyst, suppuration, and rupture are relatively much more frequent than apart from pregnancy. It is sometimes asserted that the growth of ovarian tumours is accelerated during gestation, but there is no evidence to show that pregnancy exercises any uniform influence in this direction. Still less do the facts support the view advanced by Wernich that pregnancy causes malignant degeneration in previously benign growths.

The symptoms to which an ovarian tumour gives rise in the course of pregnancy are extremely variable, and depend largely on the

presence or absence of complications. In many cases there are no symptoms beyond those of pregnancy, and from first to last there may be nothing to suggest to the woman the existence of a dangerous complication. More often, however, a careful inquiry will elicit some unusual features. While there may be simply an exaggeration of one or other of the recognised symptoms, occasional attacks of pain, of longer or shorter duration, are frequently complained of in the region of the affected ovary. Disturbances of micturition are especially common in the early months; and, in the case of large tumours, we find evidences of mechanical distension in œdema of the legs or abdominal wall, in the disturbed digestive functions, or in an embarrassment of the respiration which may be so great as to demand speedy relief. But it is when complications of the tumour supervene that we get the most serious and characteristic symptoms. The clinical features of these are identical with the same attacks in the non-pregnant. Peritonitic attacks are especially common, and occurring during pregnancy should always suggest the possibility of ovarian disease.

The recognition of an ovarian tumour during pregnancy presents, as a rule, no great difficulty, and must rest on the usual physical signs. On inspection a double tumour will often be observed, and this will be readily confirmed by palpation and bimanual examination. It is where the tumour is very large that the difficulties are greatest. The abdominal signs of pregnancy may then be entirely obscured; and even within a few weeks of term it may be impossible to feel the fœtal parts or to hear the fœtal heart sounds. It will often be possible, however, to make out the characteristic uterine contractions. An important aid to diagnosis is to be found in the method employed by Schroeder. During bimanual examination he fixes the cervix uteri with a volsellum, which is manipulated by an assistant, and thus helps in the differentiation of the two tumours.

Many mistakes in the diagnosis of this complication have been made from not attaching sufficient weight to the signs and symptoms of pregnancy which were present. In a patient with ovarian tumour suppression of menstruation should always suggest pregnancy. In the absence of abdominal signs the various symptoms, and the vaginal signs, more especially the characteristic changes in the cervix, have an increased importance.

The association of pregnancy with an ovarian tumour will always be a cause for anxiety. Though many cases pass through pregnancy and the puerperal period without accident or even without inconvenience, experience shows that this is exceptional, and that "there is no security against some formidable catastrophe." In a large series of cases collected by the writer the maternal mortality was nearly 20 per cent, and even this would have been much higher but for operation undertaken on the development of urgent symptoms. The danger lies in the possibility of complications; and these may be anticipated, in more or less severe form, during pregnancy or the puerperal period in nearly every second case. The most common complication of the tumour is torsion of its pedicle, which is met with in over 12 per cent of the cases.

Treatment.—Within recent years the treatment of ovarian disease in pregnancy has undergone great change. Till lately it was the practice to avoid interference in the absence of any urgent symptom; and where remedial measures were demanded, either to aspirate the tumour or to induce premature labour. These methods are now discarded or reserved for exceptional cases; and it is generally held that the best treatment, when an ovarian tumour is found to complicate pregnancy, is immediate ovariotomy. It can be shown that ovariotomy gives better immediate results, for mother and child as well, than either non-interference or aspiration of the cyst, while at the same time it relieves the woman of her disease. Though occasionally it may be more difficult, ovariotomy in pregnancy is in no way more hazardous than apart from pregnancy. The mortality in a large series of cases, including those fatal cases in which some complication of the tumour preceded operation, was only 3 per cent, while pregnancy remained undisturbed in over 70 per cent. The more recent cases show even better results.

There is a disposition among many, even of those who in general advocate ovariotomy, to make an exception in the case of small tumours, which they believe may be safely treated expectantly, but it should be noted that dangerous complications are quite as frequent in the small tumours as in those of larger size.

As to the time at which ovariotomy may be most safely performed, Dsirne concluded, from an analysis of 135 cases, that the second, third, and fourth months gave the best results for the mother, and the third and fourth months for the child. A larger survey shows that the operation may be performed with almost equal safety for mother and child at any period of gestation. There is, in fact, no elective period for ovariotomy in pregnancy. Whenever an ovarian tumour is detected during pregnancy, no matter how small, the rule should be to advise its immediate removal.

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The affections and accidental complications of pregnancy will naturally include: (A) those which can be distinctly traced to the presence of the gestation, and which may be one of three classes: (α) Direct results of sympathetic irritation, as sickness; neuroses, or pathological exaggerations of the physiological conditions of pregnancy; (β) Mechanical results of pressure, as from some uterine displacement; (γ) Complex, *i.e.* partly neurotic, partly mechanical, and partly obscure nutrition changes produced by the pregnant state.

(B) Those which may arise apart from pregnancy or are grafted upon or are independent of it, but which seriously interfere in many cases with its normal course, *e.g.* the specific fevers.

It is obviously impossible to classify each symptom or disease under one of the above headings, and it has been deemed preferable to give them in detail under the various "systems" involved.

I. The Nervous System.—Pregnancy intensifies the susceptibility of the nervous system; all parts of it are liable to be affected. Thus, in considering the special senses we may meet with amblyopia (apart from nephritis), hemeralopia, deafness, perversion of taste and smell, and loss of one or both. In the psychic state we meet with uncertainty of temper, melancholia, and insomnia. The motor tracts are rarely involved. Among vaso-motor disturbances there occur neuralgias, vertigo, syncope, local anæsthesia, paresis. There are also the reflex symptoms of hiccough and cough. All of these complications are as a rule less noticeable during the second half of pregnancy than during the earlier portion. *Locomotor ataxy*, *progressive muscular atrophy*, and *general paralysis of the insane* may occasionally complicate pregnancy, but so far as can be ascertained from the few cases reported nothing abnormal in the course of the gestation has been observed.

Convulsions may be due to epilepsy, hysteria, or albuminuria (*see* "Eclampsia" and "Nephritis"). Epileptic seizures are not attended by any interference with the course of pregnancy. It is said that the fits may arise for the first time during this condition, but this is doubtful; there is no albuminuria.

Among the more important instances of *paralysis* are paraplegia, hemiplegia, and paralysis of the face and of the nerves of special sense. Many of these are complicated by albuminuria. Under these conditions it is generally advisable, if the albumin does not disappear with ordinary treatment, to induce labour. On the other hand, all of them may be due to affections unaccompanied by albuminuria, such as in cerebral and spinal hæmorrhages.

Chorea is a grave complication. In the majority of cases the disease existed during childhood or at puberty. It is usually bilateral. Primigravidæ appear to be much more frequently attacked than multiparæ. It does not, as a rule, complicate subsequent pregnancies. Buist places the mortality very high, one in five proving fatal. In addition to this there is certainly a tendency to a permanent resulting disturbance. None of the usual drugs appear to have much effect in ameliorating the condition, and the obvious treatment in a large number of cases, especially if the movements are tending to increase, is artificial interruption of the pregnancy.

Tetany (tetanilla) is a tonic, usually painful spasm, beginning in and often limited to the muscles of the extremities, chiefly the hands. It is always symmetrical. No loss of consciousness has been observed. The attacks are "intermittent and of short duration (five to fifteen minutes)"; they may recur daily. The spasms are preceded by tingling or numbness in the parts presently to be affected, and the same sensations follow the relaxation of the spasm. It is commoner in delicate than in robust women. There is no pyrexia, and it may be looked upon as a slight ailment tending always to spontaneous recovery.

Tetanus is as rare as tetany. "The spasm begins in the muscles of the face and neck (trismus) and advances centrifugally, with opisthotonos." The spasm is constant, and subject to exacerbations. The temperature is usually raised, and the disease practically always terminates fatally. Robust patients are most frequently attacked. Both of these conditions must be diagnosed from hysteria and spinal meningitis.

II. Digestive and Sympathetic Disturbances.—A certain amount of nausea and vomiting of a glairy, frothy material, especially in the early morning on first assuming the upright position, is the rule in the early months of pregnancy. The nausea has been compared to sea-sickness. This usually ceases as the uterus rises out of the pelvis, and remains absent for the remainder of the gestation period. In some cases the vomiting of at least one meal a day may persist throughout the nine months without any bad constitutional effect arising beyond slight wasting. Under certain conditions, fortunately rarely, graver symptoms arise, all nourishment is rejected, and as a consequence marked emaciation sets in, the aspect of the patient becomes pinched and anxious, the tongue dry and brown; the temperature and pulse gradually rise, and she may gradually pass into a state of delirium and

die unless relieved by spontaneous abortion or artificial interruption of the pregnancy. This condition is usually called the "uncontrollable vomiting of pregnancy." Occasionally it is discovered that a retroverted incarcerated uterus is the cause of all the distress. Treatment of the minor cases of sickness is sufficiently simple. The patient should be directed to keep the bowels regular daily by means of a cascara and belladonna pill, to take simple and digestible food, and to drink a cup of tea and eat a rusk before rising in the morning. An effervescing mixture, with hydrocyanic acid, thrice daily is as a rule the only medical treatment required. If the vomiting, however, persists various other remedies have been suggested, such as oxalate of cerium (gr. v.) in pill three times daily, as recommended by Simpson, small doses of morphia, or hydrochlorate of cocaine. Should these fail it may be necessary to put the patient to bed and feed her entirely on nutrient enemata, the thirst being relieved by the sipping of hot water. In severe cases certain local applications have been applied to the cervix with considerable benefit. The author has been accustomed to apply a solution of cocaine or strong tincture of iodine through a speculum by means of a camel's-hair brush. In some cases this has been followed by almost instantaneous relief, in others has absolutely failed. Should the uterus be retroverted and impacted it is best to draw off the urine, push the fundus above the pelvic brim, and support it there by means of a watch-spring ring pessary. Chapman recommends the application of spinal ice-bags in intractable cases, but no great success seems to have attended this method. Copeman advocated the dilatation of the cervix with the finger, and stated that he met with many favourable results from the adoption of this procedure. It is obvious, however, that it must only be resorted to with the greatest caution, as a miscarriage may easily be induced.

Should the vomiting persist in spite of the above-mentioned remedies, nothing remains but to procure abortion; this should not be too long delayed, and should only be carried out after the practitioner's opinion has been fortified by another authority. The introduction of one or more laminaria tents is the better plan, although the completion of the operation at one sitting by means of Hegar's dilators and the curette has been strongly advocated by many obstetricians. Unfortunately, even this does not always save the patient, more especially if interference has been unnecessarily delayed.

Minor forms of digestive disturbance which may be mentioned are capricious appetite (pregnancy longings), toothache, heartburn, and flatulence.

Constipation is the rule in the pregnant woman, more especially in the earlier months if the patient is naturally of a constipated habit; it is probably due to interference with the venous circulation and interchange of fluids, later to pressure of the uterus. Unless great care be adopted the colon may become filled with hard scybala, which may remain there throughout the pregnancy, and only give rise to trouble after parturition has taken place. Purgation produces a diarrhœa which may easily deceive both practitioner and patient without evacuating the contents of the distended colon.

Ptyalism is a very distressing complication, and although, as a rule, the flow of saliva is not excessive, in some marked cases it may amount to at least a quart in the twenty-four hours. The patient may find it necessary to spend her time catching the saliva which flows from her mouth into a bowl. The salivary glands are often enlarged and tender. No remedy but the termination of pregnancy seems to be of any avail, but astringent gargles with counter-irritation over the parotid areas may be tried.

Jaundice (icterus) may be due (*a*) to catarrh of the bile-ducts; (*b*) to acute yellow atrophy; (*c*) malignant disease. Icterus should always be looked upon with a certain amount of apprehension when complicating pregnancy, and, so long as it runs the usual course, no special treatment is necessary; should it, however, persist, the patient may be the subject of acute atrophy or possibly malignant disease. The former consists in "poisoning of the system by an accumulation of bile-acids in the blood, together with secondary degeneration of the parenchyma of the great glands and of the substance of the muscles, especially of the heart." A diagnosis must, of course, be made from acute phosphorus poisoning. The prognosis is very grave as regards both mother and the fœtus. The question of induction of abortion should usually be decided in the negative. Carcinoma is only recognised with difficulty during pregnancy, but it is quite obvious after the uterus has been emptied of its contents. Induction of premature labour may be necessary to relieve the intense pain which frequently accompanies this condition.

Diabetes as a disorder of the hepatic glycogenic function has been inserted here; it must not be confused with physiological glycosuria, which is frequently present in normal pregnancies. It is, fortunately, a rare condition, and we owe to Matthews Duncan any knowledge there is on the subject. He reported twenty-two cases, of which four ended fatally during the puerperium from collapse or coma. He concludes from his researches into the histories of published cases that diabetes may come on during pregnancy, and that it may cease with the termination of that

condition. Diabetics are not necessarily sterile, but pregnancy is very liable to be interrupted in its course, and nearly always in consequence of fœtal death.

Diabetes insipidus has been noted in many cases of pregnancy. The amount of urine passed may reach as much as 300 ounces in 24 hours. It appears to set in about the fifth or sixth month, and to disappear entirely after labour.

III. Cardio-vascular Disturbances.—During pregnancy the work of the heart is augmented owing to the larger quantity of blood and the increased resistance in its circulation. During the later months this task is added to in consequence of the decreased power of expansion of the thorax and the compression of the large abdominal veins by the pregnant uterus. In spite of this additional work, a healthy heart is possessed of so much reserve force that hypertrophy is unnecessary. Larcher affirms that in all cases of pregnancy there is hypertrophy of the left ventricle. Should the balance between the heart's beat and the increased amount of blood be disturbed we meet with such complications as palpitation, temporary vertigo, faintness, and dyspnœa: these need give rise to no anxiety provided the heart muscle and valves are sound.

The pregnant woman may be the subject of several *forms of cardiac lesion.* They are: (1) Adhesions from former pericarditis, among which would be included those in which the heart has been displaced by pleural adhesions; (2) Myocarditis and degeneration of the cardiac muscle; (3) Endocarditis, chiefly affecting the valves (valvular heart disease); and (4) Acute endocarditis grafted upon the chronic form. It is important to bear in mind that valvular disease is not the only form of cardiac disorder which may give trouble: all the above-mentioned conditions may produce much distress and danger when complicating pregnancy, and it is frequently less often owing to a disturbed valvular mechanism than to a loss of reserve force of the heart that the so-called compensatory disturbances arise. When compensation becomes insufficient during pregnancy certain phenomena occur in greater or less severity: these are varicosities of the legs and labia, œdema of the feet, legs, and vulva, or even general anasarca, palpitation, and syncope, irritable cough, bronchitis with dyspnœa and cyanosis, hæmoptysis, albuminuria, and, lastly, serous effusions into the peritoneal and pleural cavities. Aortic stenosis is certainly the most dangerous form of valvular lesion; but many women with marked mitral lesions may go through pregnancy and labour without untoward symptoms arising, provided compensation be present. Abortion and premature labour occur frequently, and it is proved by statistics that fatal results are nearly twice as frequent in cardiopathic multiparæ as in primigravidæ.

Treatment in mild cases should be confined to the avoidance of over-fatigue, too much stair-work, and the regulation of the bowels, straining at stool being especially avoided. Compensatory disturbances may appear as early as the ninth week of pregnancy, though as a rule not until the fifth month. Should albuminuria set in it must be looked upon as a serious phenomenon.

As a rule a mixture of liquor arsenicalis, ℥v., and liquor ferri dialysati ℥x., or liquor strychniæ ℥v. thrice daily, will counteract the anæmia and want of tone in these cases; a stimulating mixture of ether and ammonia will be useful to cope with the attacks of syncope.

Should marked œdema or effusion set in with dyspnœa, the exhibition of digitalis in the form of tincture or infusion will be indicated, more especially where the lesion is valvular and of the mitral variety; it is contra-indicated in aortic disease. Tincture of strophanthus (℥iii.-℥x.) is in some cases an extremely valuable heart tonic, but its administration is also not advisable in aortic stenosis. Treatment by induction of abortion may be attended by very grave results. Schlager reports three cases in which he resorted to induction of premature labour, all of which terminated fatally. We must consider that the condition of any patient in which it is thought necessary is one of extreme gravity, and the mere fact of interrupting the pregnancy will not stay the cardiac degeneration which is going on.

Varicose veins are most often observed in multiparæ, although they are not uncommon in primigravidæ. They may be capillary in form, "in which case an arborescent network of bluish vessels is seen round the ankles, and especially on the inner surface of the thighs." The deep veins lying below the point of division of the iliac veins are also involved, as is shown by the constant occurrence of hæmorrhoids and vulval varicocele. The left limb is affected more than the right, and more towards the latter end of pregnancy.

The chief danger in connection with these varicosities is the possibility of *thrombosis and embolism.* Spiegelberg has seen four fatal cases of this kind. Another danger is that by ulceration or traumatism one of the veins may rupture and profuse hæmorrhage follow. This is especially the case in the vulva, where the accidental rupture of a plexus by a broken utensil has been known to lead to fatal bleeding.

Hæmorrhoids are probably the most frequent and distressing complications of advanced pregnancy; treatment should be upon the same lines as in the non-gravid state, and requires no further notice here.

Thyroid enlargement is very usual to a slight extent, and was observed by ancient writers; it is, as a rule, a purely vascular change, and unless there is a true goitre present, disappears after the termination of the pregnancy.

IV. Blood Changes. — The blood-mass or actual quantity of blood is markedly increased during gestation, and more especially in the later months, producing a spurious plethora. Its composition is, however, very radically changed, and becomes more watery, the albumin is deficient in the serum, and the red corpuscles are diminished in number. In addition to this the fibrinous element is much increased, as are urea and extractives, especially after the sixth month. Hence the blood condition is really one of anæmia rather than plethora. It has been shown by Wilcocks that the blood of pregnancy differs from chlorosis in that the individual blood cells are not impoverished in the former, "but are lessened in comparative number owing to an increase in the water of the plasma, the result of the progressive enlargement of the vascular area during pregnancy." In both conditions the hæmoglobin is lessened.

This watery state of the blood may be much exaggerated under certain circumstances, giving rise to what is called (*a*) *hydræmia* or serous plethora; it consists of serous effusion into the cellular tissue and peritoneal, pleural, and pericardial cavities. The œdema may occasionally form very rapidly, and, if in the thorax, is attended by great danger. The lower extremities are also frequently affected, but the face, as a rule, remains free, and there is no albumin. This œdema must be carefully distinguished from the ordinary puffiness of the feet and legs which is so frequent in the later months of normal pregnancy.

(*β*) The second type to which this poverty of blood may lead is *progressive pernicious anæmia*. In this condition too few red corpuscles are produced, and too many shrunken and ill-developed ones are thrown into the circulation. Fatty degeneration of the heart, especially of the papillæ, follows, and a somewhat similar change in the minor coats of the arteries and capillaries. The disease begins very insidiously during the early months, but the graver symptoms do not manifest themselves until later; the prognosis is extremely grave. Of Graefe's 25 cases almost all died. Abortion frequently occurs; induction of labour is absolutely indicated, and as early as a diagnosis is made. Transfusion has been tried, but has been so far attended by uniformly fatal results. The treatment of extreme cases of anæmia must be dietetic and medicinal. The diet should be easily assimilated, and consists of milk, eggs, and beef tea, while iron in all forms and combined with arsenic or phosphorus is strongly indicated.

Purpura hæmorrhagica is defined by Immermann as an acquired and transitional diathesis which suddenly and unexpectedly, and always sporadically, occurs in persons who have been apparently quite healthy, and which cannot be traced to family taint or history as in hæmophilia. The prognosis is extremely serious when complicating pregnancy, the large majority proving fatal. Abortion or premature labour almost inevitably takes place, but at variable periods, either owing to the serious general disturbance or to hæmorrhage into the placenta. The disease is not transmitted, so far as has been observed, to the fœtus; it is certainly one of the causes of intra-uterine death.

V. Syphilis. — If infection be recent this disease is followed by the usual primary symptoms; the secondary and tertiary signs are usually of a mild type, probably due to the more active proccsses of nutrition in the pregnant condition.

Constitutional syphilis has a very injurious influence on the fœtus; intra-uterine death may occur, followed by abortion, or if the child is born alive it is either premature or very weakly, and may die early. Should it appear healthy at birth congenital symptoms speedily set in, taking the form of marasmus, nasal ozæna, corneitis, rashes, and characteristic dentition.

The placenta may be affected in two ways: either a general fibrosis may invade its substance, or distinct gummata may be formed.

A woman who has given birth to a syphilitic child may, according to Colles, expose herself to infection by suckling it or otherwise without risk.

Treatment during pregnancy should always be systematically carried out; mercury and iron, combined with a little opium, being given in the form of pill thrice daily throughout the gestation period.

VI. Pulmonary Disturbances.—There seems to be a slight increase in the amount of carbonic acid discharged from the lungs during pregnancy. During the later months there is a diminution in the vital capacity of the lungs, the thorax being increased in breadth, but diminished in its antero-posterior diameters—this being exactly reversed after the termination of labour. The changes are due probably "to the diaphragmatic points of insertion being pulled upon by the descending uterus; the base of the thorax yields at its weakest spot, *i.e.* the anterior wall, the flexible costal cartilage being attached there." The result of this is that the sternum recedes, thus reducing the antero-posterior thoracic diameter, while the ribs become increased in their lateral curves. These physical changes will account for the feeling of oppression felt by the patient towards the end of pregnancy, and the relief obtained by the "lightening before labour" when the uterus sinks deeply into the pelvic brim.

Among acute pulmonary diseases *pneumonia*, although extremely rare in the pregnant woman, is markedly fatal. The gestation may be interrupted, but this is more likely to occur in the later months; this is probably due to the incomplete aeration of the maternal blood, and partly to the death of the fœtus, which is the

result of the high temperature. Double pneumonia is always fatal, and septic processes are found to be much more frequent sequels to labour than in normal cases. Induction of premature labour is contra-indicated, all treatment being directed to the pulmonary lesion.

Phthisis does not necessarily lead to sterility; the effect of pregnancy upon this lesion was formerly thought to be favourable, but at present the reverse seems to be the generally accepted opinion. As to whether this condition favours the growth of tubercle or not is a little undecided. Treatment by artificial abortion is strongly contra-indicated, and the induction of premature labour is only admissible when the enlarged uterus interferes mechanically with the expansion of the lungs.

Bronchitis of the chronic type leads to much dyspnœa and distress during pregnancy, and the engorgement of the right heart may become so marked that venesection or leeching may be necessary; it is frequently followed by great and immediate relief.

Pleurisy leading to effusion or empyæma must be treated after the same methods, surgical and otherwise, as would be followed if the disease attacked the unimpregnated female. (*See* "Pleura.")

VII. Renal Affections.—In the kidneys of the pregnant woman hyperæmia and increased epithelial proliferation are found, and cannot be attributed to the result of the pressure of the enlarged uterus. The urine is somewhat altered; it may be acid, rarely alkaline. As pregnancy advances, phosphates, sulphates, urea, and uric acid decrease, while water and chlorides increase; the marked watery condition of the urine is due to increased arterial tension. To this latter factor the presence of transient or permanent albuminuria is due. There seems little doubt that vesical hæmorrhage or catarrh of the mucous membrane may occur, leading to a temporary appearance of albumin. This condition always causes anxiety, and the urine should be carefully examined at regular intervals throughout the pregnancy.

Albuminuria is present in a large number of cases of puerperal eclampsia, and also in certain forms of paralysis of the special nerves (amaurosis), or of the spinal system; it may also occur in puerperal mania. (*See* "Puerperium, Puerperal Insanities.")

The presence of albumin to a small extent is found in a considerable proportion of pregnant women (2 to 4 per cent), but it usually rapidly disappears after labour, which may be concluded without any untoward symptoms arising. Care must be taken to distinguish between the patient who is the subject of chronic renal disease and the one in whom the albumin has appeared for the first time during pregnancy.

The causation of this complication is still the subject of much discussion; but there are four theories which should be considered, none of which, however, seem to be entirely adequate: (1) Pressure on the venous renal vessels by the uterus: this view is favoured by the fact that albumin rarely appears until after the fifth month of pregnancy, and is much more common in primigravidæ, owing probably to the resistance of the abdominal parietes being greater. On the other hand, the albumin appears long before the uterus is large enough to exert any pressure upon the renal veins, and large growths (fibro-myomata and ovarian cytomata) do not always lead to this condition; there must obviously be something in addition to the uterine pressure. (2) That the ureters are obstructed or at least narrowed by the pressure of the gravid uterus. This has been proved by experiment to be untenable, and no constant dilatation of the pelvis of the kidney nor of the ureters themselves has ever been observed. (3) That it is due to the increased work thrown upon the kidneys by the addition to the blood of the fœtal waste products. (4) That too much stress has been laid upon the mechanical causation, and that the process is rather due to the presence of some toxic material in the blood (Clifford Allbutt).

If the albuminuria is recent, symptoms as a rule do not show themselves before the seventh month; they may then come on very suddenly —persistent headache, vertigo, diplopia, vomiting, and slight œdema of the face, hands, and feet. A working-woman will often complain of inability to thread her needle. The albuminuric deposit consists of para-globulin; in chronic cases the deposit is mainly serum-albumin. Various paralyses may arise, and well-defined ocular changes (*see* "Nephritis"); œdema of the lungs may also set in. There will be a decided tendency to hæmorrhages and to abortion, which latter is owing to the death of the fœtus; it may be expelled or be retained in a macerated condition. Fœtal death is probably due to imperfect nutrition or to changes in composition of the maternal blood. Eclampsia is more likely to occur in recent cases, while ocular changes are usually present in the more chronic forms.

Treatment varies according to the period at which the albuminuria first appears. When it begins early the object of the practitioner should be to promote an increased secretion of urine, and to produce diaphoresis and purgation. Saline diuretics, such as acetate and citrate of potash, fulfil the first requirement, while watery motions will result from daily administration of compound jalap powder. The hot air bath and warm wet pack produce copious sweating and are of great assistance. The patient's urine should be examined every week, and if, in spite of the above remedies and a milk diet, the albumin still increases, absolute rest in bed must be enforced. By some authors milk diet is prescribed, the milk being skimmed. Should

these measures fail, the question of induction of abortion must be seriously considered. It is in itself attended by the risk of bringing on an eclamptic attack; but considering the matter from all points, and if severe headache and dizziness are present, there is no doubt of the propriety of performing it. The operation may be carried out by means of tents, or at one sitting under an anæsthetic.

If the albumin appears late in the progress of gestation active measures are at once necessary —free purgation, administration of chloral and bromide of potash, and everything prepared for the induction of premature labour. (*See* "Eclampsia," vol. iii. p. 3.)

VIII. Cutaneous Rashes.—*Anomalous pigmentation* consists of the deposit of a brownish pigment in various sites on the skin, especially the face, areolæ, and external genitalia. The forehead is covered with dark, irregular patches more marked near the roots of the hair; dark rings appear round the eyes and occasionally round the root of the nose and the mouth (pregnancy mask). In cicatricial tissue the pigmentation is often very marked. Similar staining is found about the navel and the umbilico-pubic line. The thenar and hypothenar eminences may become stained a deep pink colour immediately pregnancy commences; this variety disappears immediately after labour, but a slight amount of pigmentation is always permanent to some extent.

Herpes gestationis usually begins in the form of clusters of vesicles on the vulva or on the extremities, and may spread over the whole body. The rash persists as a rule throughout the pregnancy, and disappears spontaneously after delivery, returning with a fresh conception. The treatment is exactly as in the non-gravid condition.

Impetigo herpetiformis appears as small pustules on the inner side of the thighs. They spread rapidly and may involve the scalp. Much constitutional effect is produced, and the disease may even run a fatal course. (*See* "Dermatitis," vol. ii. p. 304.)

Pruritus, or itching, may affect the vulva only or the whole of the body surface. It may become so intense, and produce such constitutional disturbance as to lead to a consideration of induction of premature labour.

It is usually a neurosis, but may be due to eczema or intertrigo. The best modes of treatment are purgation and the administration of the bromides, with local cleanliness and the application of lotio nigra or a solution of hydrochlorate of cocaine. Sitting in a hot bath containing boracic acid just before going to bed is often a very effectual remedy.

IX. Specific Fevers.—It was formerly believed that the pregnant woman was protected or immune from the specific fevers, and that the pregnant condition had a curative effect on others; this has been proved to be entirely erroneous.

Many of the fevers when complicating pregnancy may run an unaltered course, or may produce grave complications. Abortion often results from the high temperature, or from the effect of the diseased maternal blood upon the epithelium of the fœtal placenta. The fœtus may be born with the rash fully out upon it. It is said that the child born of a woman suffering from a specific fever may in some instances be immune from that particular variety.

(*a*) *Variola.*—This is the most frequently observed among the exanthemata attacking the pregnant woman; it is attended by extreme danger, owing to the liability of the patient to the hæmorrhagic variety; premature labour and abortion are hence very common, and severe uterine hæmorrhage frequent. A peculiar clinical fact has been noted, that in smallpox epidemics many women will abort with much hæmorrhage without the rash appearing. It is always advisable to vaccinate a pregnant woman if she is liable to be brought into contact with this disease. The mortality in the severer forms is at least 90 per cent, while in the milder epidemics about 10 per cent. (*b*) *Enterica* (typhoid) is said to be more frequent during the earlier months of pregnancy than later; abortion and premature labour occur in nearly 70 per cent. The higher the temperature the greater the risk of abortion. The use of cold baths to reduce the fever certainly conduces to a premature interruption of pregnancy. The prognosis is good in mild cases occurring early in gestation, but more grave in the later months and after a continuance of pyrexia. The treatment cannot in any way be modified from that pursued in the non-pregnant state. (*c*) *Scarlatina* is more frequent after delivery than during the pregnant condition, leading to the supposition that the patient may to some extent be immune. (*d*) *Morbilli*, unless of a very grave type, runs a normal course and without affecting mother or child. (*e*) *Erysipelas* is dangerous from the probabilities of postpartum infection taking place, and acute septic disease setting in indistinguishable from puerperal septicæmia. (*f*) *Continued fevers* may attack the pregnant woman, and from the pyrexia lead to abortion.

X. Osseous Changes.—Osteophytes are small bony deposits which form on the inner tube of the cranial vault, and chiefly on the frontal and parietal bones. They mainly lie along the groove of the middle meningeal artery. It is said that they are, however, not entirely confined to the pregnant condition. (*See also* "Pregnancy, Physiology of," p. 222.)

Osteo-malacia (malacosteon) is an extremely rare complication of pregnancy in the British Isles, but is common in Germany and Austria.

This disease may be defined as a degeneration of the bones, chiefly of the pelvis and spine, resulting in the loss of lime salts, and consequently a softening: it is entirely dependent upon the pregnant state. No treatment seems of much avail while the pregnancy goes on, except the removal of the ovaries and tubes with or without the pregnant uterus (Porro's operation). Should the disease continue after labour has terminated, oöphorectomy is advisable and is usually successful.

XI. Vesical Complications. — The bladder becomes more irritable, the desire to micturate more frequently setting in soon after pregnancy commences; this is probably due to the anteversion which occurs at this period. This may go on until labour is terminated. Incontinence and absolute retention may result from pressure of the cervix on the neck of the bladder with an impacted retroverted uterus. This latter complication, which is not infrequently attended by a dribbling (overflow from much distension), is relieved in milder cases by the passage of the catheter; the uterus then gradually rises above the pelvic brim, and all danger is at an end. In more severe cases an anæsthetic is necessary, and manual reposition of the displaced uterus necessary, the left hand being passed into the vagina, or two fingers into the rectum; a stiff ring pessary should be inserted to keep the fundus in its place. Under very rare circumstances puncture of the fundus uteri per vaginam by means of a trocar is necessary; the liquor amnii escapes, and the uterus being reduced in bulk can then be pushed up out of the pelvis—abortion, of course, being the inevitable consequence.

Cystocele, or prolapse of the anterior vaginal and posterior bladder walls, becomes more troublesome during pregnancy, owing to the increased congestion of the mucous membrane, and therefore slightly more protrusion at the vulval orifice. (*See also* "Bladder, Diseases of," vol. i. p. 384, and "Catheter and Septic Infection of the Urinary Tract," vol. ii. p. 73.)

XII. Mental Complications. — Insanity of pregnancy is much less common than that attacking the puerperal or nursing woman (about 12 per cent). Many women are naturally despondent and low-spirited during gestation, and the form of insanity usually observed is that of melancholia. It affects primigravidæ more frequently than multiparæ, and is commoner in women between thirty and forty years of age than in younger ones. Hereditary taint is a very important cause, as also are epilepsy and alcoholism in one or both parents. The disease may begin with conception, or more usually about the third or fourth month, and may recur with each successive pregnancy. The tendency is usually suicidal, but may be homicidal, or rarely a disposition to kleptomania. The prognosis is favourable, and the disease frequently disappears after labour. In bad cases the question of induction of abortion may have to be considered. In all such cases the patient should not be left alone, but should have a cheerful, firm nurse in constant attendance upon her. Change of scene without excitement is of the first importance. (*See also* "Puerperium, Puerperal Insanities.")

Causes of Death during Pregnancy

These may be divided into three groups—(1) Where death is due to some cause which is not connected with the pregnant state; (2) where death is a consequence or result of pregnancy; (3) where a pathological condition existed before the onset of or developed during pregnancy, and which has been so aggravated by that state as to result in the patient's death. The first group requires no further consideration here, as it belongs to the domain of general medicine and surgery. Under the second heading two types require consideration; in one where death results from a normal, in the other from an abnormal, pregnancy. Normal pregnancy of itself very rarely causes death during its course, but it may under the following conditions:—Excessive vomiting, which is frequently associated with some pathological condition such as hydramnios, myxomatous degeneration of the chorion, albuminuria, etc., may occur without any such accompaniments, and, if allowed to continue, may lead to fever, syncope, delirium, coma, and, finally, to the death of the patient. Chorea sometimes develops during pregnancy, and, it is said, causes a mortality of about 1 in 5 cases. Spontaneous rupture of an apparently healthy uterus has been recorded as a cause of death. The development of varicose veins in the legs, pudenda, and pelvis as a result of pressure from the growing uterus is frequently met with. Phlebitis may follow, and death due to the clotting of blood in the pulmonary artery and its branches. Again, rupture of varicose veins of the legs or vulva has produced such profuse hæmorrhage in some cases as to kill the patients. Pregnancy sometimes so profoundly affects the blood as to lead to hydræmia or serous plethora, or to pernicious anæmia. Œdema of the lungs may occur in the former condition with a fatal result. Prognosis in pernicious anæmia is very serious. Eclampsia is one of the commonest causes of death during pregnancy, but this has been fully treated of under its appropriate heading. Acute yellow atrophy has a curious relationship to pregnancy, and terminates fatally practically in all cases within six days. Lastly, death may result from manipulations, as in a case recorded by Hektoen, where air was admitted into the uterine veins from the improper administration of a vaginal douche, and in cases of procured abortion from sepsis. In abnormal pregnancies deaths are more frequently met

with, and of all causes rupture of an ectopic gestation is the commonest, the fatal termination being produced by shock and large intra-peritoneal hæmorrhages. Myxoma of the chorion may produce a similar catastrophe, either by causing perforation of the uterus or hyperemesis gravidarum. The latter may also be a sequela of hydramnios. Retroversion of the gravid uterus, if not rectified by nature or the obstetrician, may lead to sloughing of the bladder or other pelvic viscera, or to uræmia, and in either condition death of the patient may result. As to cases where a pathological condition existed before the onset of, or developed during pregnancy, and which has been so aggravated by that state as to result in the patient's death, little need be said here, as the subject has been dealt with fully in the preceding pages. Of all pre-existing conditions heart diseases, whether of the valves or the myocardium, are most unfavourably influenced by pregnancy. This holds specially true in cases of mitral stenosis and aortic incompetence, though even in these cases a fatal termination is much more common during labour than during pregnancy.

Hæmorrhage

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ANTEPARTUM HÆMORRHAGES

ARISING AS A RESULT OF THE PREGNANCY

Antepartum hæmorrhages arising as a direct result of the pregnancy may be divided into three main groups:—

A. Hæmorrhages occurring during the first three months of pregnancy.

B. Hæmorrhages occurring during the second three months of pregnancy.

C. Hæmorrhages occurring during the last four months.

In adopting this classification, pregnancy is considered as the duration of ten lunar months of four weeks each. The first group includes hæmorrhages occurring before the full formation of the placenta. The second group includes hæmorrhages occurring from the time the placenta is formed to the time the fœtus becomes viable. And the last group includes hæmorrhages occurring after the fœtus is viable.

A. HÆMORRHAGES OCCURRING DURING THE FIRST THREE MONTHS

The hæmorrhages of the first three months of pregnancy, which arise as a direct result of the pregnancy, have three chief causes:—

(1) ABORTION.—Abortion is the term applied to the expulsion of the ovum from the uterus before the full formation of the placenta, that is before the end of the third month. It is a process which is of necessity accompanied by hæmorrhage, as it implies the rupture of the vascular adhesions which connect almost the entire surface of the ovum with the decidua. The varieties, or perhaps it would be more correct to say the phases, of abortion are as follows:—

(*a*) *Threatened abortion, i.e.* the onset of pain, hæmorrhage, and a varying degree of dilatation of the cervix occurring in a patient during the first three months of pregnancy.

(*b*) *Cervical abortion, i.e.* the condition which occurs when the ovum is expelled into the cervical canal, in which it becomes incarcerated as a result of the failure of the external os to dilate.

(*c*) *Missed abortion, i.e.* the retention of the ovum in the uterine cavity after its death has occurred.

(*d*) *Complete abortion, i.e.* the expulsion of the entire ovum.

(*e*) *Incomplete abortion, i.e.* the expulsion of a part of the ovum, the remainder being retained in the uterus. (*See* "ABORTION," vol. i.)

(2) ECTOPIC GESTATION.—Ectopic gestation, or extra-uterine pregnancy, is the term applied to the development of the ovum outside the cavity of the uterus. Such development may take place primarily in either the tube or ovary, but practically it always occurs in the former. As a hæmorrhage-producing factor, it is met with in two phases during the first three months—

(*a*) *Before primary rupture of the gestation sac, i.e.* while the ovum is still contained in the intact tube or ovary, as the case may be.

(*b*) *At the time of primary rupture of the gestation sac*, an event which occurs as a rule between the sixth and tenth weeks. (*See* "Ectopic Gestation," vol. iii.)

(3) Myxoma Chorii.—Myxoma chorii, or hydatidiform mole, is the term applied to a myxomatous degeneration which attacks the chorionic villi in certain cases. As a result, the villi become changed into cysts varying in size from that of a pin's head to that of a grape. If, as usually happens, the greater part of the ovum is affected by this change, the fœtus dies and is absorbed. If, however, only a small part of the ovum is diseased, the fœtus may continue to develop. The myxomatous change always commences before the end of the third month, that is before the disappearance of the chorionic villi. However, hæmorrhagic discharges may occur not only during this period, but as long as the uterus remains unemptied, and accordingly it must be set down as a cause of hæmorrhage not only during the first three months, but also during the second three. It will be most convenient, however, to refer to it under the former period only.

The above are the three causes of hæmorrhage during this period. Inasmuch as each of them is fully treated in the articles "Abortion," "Ectopic Gestation," and "Moles," the writer will here confine himself to a brief account of the method of distinguishing between them.

In order to recognise the cause of hæmorrhage in any case the following points must be ascertained:—

(1) The existence of pregnancy.
(2) The duration of pregnancy.
(3) The present size of the uterus, and the nature of any alterations which may take place in it from day to day.
(4) The condition of the adnexa.
(5) The nature and duration of the discharge.
(6) The nature of any solid matter which may have been expelled from the uterus.
(7) The condition of the cervix.
(8) The symptoms of the patient.

(1) *The Existence of Pregnancy.*—The existence of pregnancy is the first point to be decided. This can be done by determining the presence of its usual subjective and objective symptoms.

(2) *The Duration of Pregnancy.*—The duration of pregnancy must be first ascertained as far as possible by the patient's history, without taking the size of the uterus into consideration. This is an important point to remember, as in some cases the diagnosis of the nature of the case will be largely based upon the difference between the actual size of the uterus and the size it ought to be in accordance with the period of pregnancy.

(3) *The present Size of the Uterus and the Nature of the Alterations which take place in it from Day to Day.*—The present size of the uterus is a considerable aid to diagnosis in cases of hæmorrhage, when it is taken in connection with the supposed date of pregnancy. If the uterus corresponds in size with the duration of pregnancy, it is strong evidence for supposing that it contains a living ovum, or one which has only quite recently died. If, however, the uterus is larger than the period of pregnancy accounts for, the possibility of myxoma chorii must be thought of. If, again, the uterus is smaller than the period of pregnancy would lead us to suppose it ought to be, it suggests, first, that perhaps the pregnancy is extra-uterine, and, secondly, if that supposition is proved to be erroneous, that the ovum is probably dead.

The alteration in size of the uterus from day to day is also important. If it increases at the natural rate, it is almost conclusive evidence that the ovum is intra-uterine and living. The only other condition under which this rate of increase could occur is in the case of a missed abortion, in which just sufficient intra-uterine hæmorrhage is taking place each day to cause the same rate of increase that a growing ovum would cause. Such a state of affairs is necessarily so rare that in practice it may be neglected. If the uterus ceases to increase in size, or even commences to become smaller, it is equally strong evidence that the ovum is dead, that is, that a condition of missed abortion is present. If the uterus grows more rapidly than is normal, it is strongly in favour of myxoma chorii.

(4) *The Condition of the Adnexa.*—The condition of the adnexa is of importance, if the question of the possibility of the existence of an extra-uterine pregnancy arises. If they are normal in size on both sides, the possibility of extra-uterine pregnancy is immediately eliminated. The typical condition met with in extra-uterine pregnancy prior to the rupture of the sac is as follows:—A globular or ovoid tumour, varying in size from a pullet's egg to an orange, is found at one or other side, corresponding in position to the tube of the same side. The tumour is elastic to touch, and slight pulsation may be felt in it owing to the increased size of the vessels which supply it. The uterus, which is also somewhat enlarged, is displaced towards the opposite side by the tumour. If rupture has occurred, it may be possible to determine the disappearance of a tumour whose existence had been previously ascertained, and the gradual formation of another tumour in Douglas' pouch.

The physical signs which help to distinguish between an unruptured tubal pregnancy and any other tumour of the adnexa are:—

(*a*) In tubal pregnancy the enlargement corresponds to the position of a tube rather than of an ovary. Ovarian pregnancy is said to occur, but it is so rare that it may be neglected.

(*b*) An extra-uterine pregnancy is usually

unilateral. Inflammatory disease of the tubes is almost always bilateral.

(*c*) The sac of a pregnancy pulsates, and occasionally contractions of its walls may be felt (Kelly).

(5) *The Nature and Duration of the Discharge.*—Slight hæmorrhagic discharge is not indicative of anything definite. It occurs in all forms of abortion, in extra-uterine pregnancy, and in myxoma chorii. Profuse hæmorrhage is conclusive evidence of some form of abortion, most probably threatened or incomplete. A profuse, watery, blood-stained discharge points strongly to myxoma chorii. If small cysts are found in it, it is pathognomonic of that condition. More or less profuse dark-coloured discharge shows that the ovum is dead, and that part or all of it is still retained in the uterus. Putrid discharge shows that the ovum is not only dead, but that decomposition has occurred. If the discharge quickly lessens, and after a few days disappears altogether, it may usually be taken to show either that the threatened expulsion of the ovum has passed off, or that the uterus has emptied itself, that is, that complete abortion has occurred.

(6) *The Nature of any Solid Matter which may have been expelled from the Uterus.*—It cannot be too strongly insisted upon, that, in the case of a patient who is bleeding, all matter which is expelled from the uterus must be most carefully examined. It is by so doing that, in the majority of cases, we obtain the necessary information to enable a diagnosis to be made. The expelled matter, if we can obtain it all, gives absolute information as to what has happened in the uterus, while the information obtained in other ways is generally more or less problematical. If an entire ovum is expelled, it is self-evident that complete abortion has taken place; or if only a portion, that the abortion is incomplete. In order to be absolutely certain that the case is one of abortion, either chorionic villi or some fragment of a fœtus must be found. A mere mass of decidua does not enable us at first sight to say whether the case is one of intra- or extra-uterine pregnancy; a microscopical examination of it will be necessary. Dakin states that the true decidua of an intra-uterine pregnancy is indistinguishable from the false decidua of extra-uterine pregnancy. However, in the former case some fragment of chorion or amnion will be found by the aid of the microscope, and this of course is pathognomonic. If intra-uterine pregnancy is excluded, the expulsion of a decidual cast of the uterus in the presence of a growing pelvic tumour is diagnostic of an extra-uterine pregnancy (Routh). As has been mentioned above, the presence of cysts in the discharge is pathognomonic of myxoma chorii.

(7) *The Condition of the Cervix.*—The condition of the cervix is of use, not only in determining whether the patient is pregnant or not, but also in distinguishing between the different forms of abortion. In a threatened abortion the cervical canal usually becomes slightly patulous. If the ovum has been detached and is in process of expulsion, it is forced against the inner os, which accordingly dilates, the external os still remaining closed. The cervix as a result becomes conical in shape, the base of the cone corresponding with the inner os, the apex with the external os (*v.* Fig. 1); in other words, the circumference of the cervix at the cervico-vaginal junction is increased. If the external os will not dilate to allow the ovum to pass through, the latter is gradually expelled into the cervical canal,

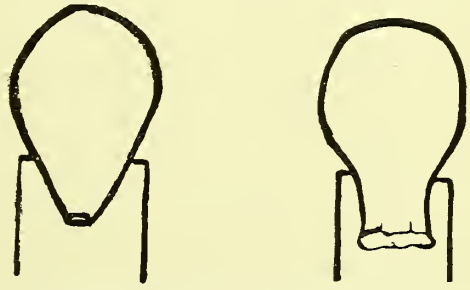


FIG. 1.—Diagram representing the shape of the cervix during, and subsequent to, the expulsion of the ovum.

which dilates to a sufficiently large size to receive it. The inner os may then partially contract again, and the ovum become incarcerated in the ballooned-out cervical canal. Thus the condition known as cervical abortion arises. If, however, the external os dilates in the usual manner and the ovum is expelled, the inner os closes again completely, while the external os remains dilated for some days. The cervix thus acquires a trumpet-shape, the mouth of the trumpet corresponding to the external os (Fig. 1). If the ovum has been completely expelled, the cervix gradually regains its normal shape; if, on the contrary, a part of the ovum is retained, complete closure of the canal rarely occurs.

(8) *The Symptoms of the Patient.*—The three chief symptoms of the patient in either abortion or extra-uterine pregnancy are hæmorrhage, pain, and collapse. In abortion, the three have usually a due relationship to one another. The degree of collapse is in proportion to the amount of hæmorrhage and pain which is occurring. The amount of pain is, however, by no means constant. In the case of a ruptured extra-uterine pregnancy, on the other hand, the first point which may strike us with regard to the case, is that the collapse is altogether out of proportion to the amount of hæmorrhage which is apparently occurring. The accompanying pain is also greater than in abortion. Such a condition should always very forcibly suggest the possibility of internal

hæmorrhage. In missed abortion, the previously existing subjective symptoms of pregnancy disappear, and are replaced by various ill-defined phenomena, the result of the absorption of ptomaines from the dead ovum.

These are the principal diagnostic phenomena to be looked for in any case of hæmorrhage during the first three months of pregnancy. It may be of use to sum them up in their relationship to the various causes of hæmorrhage.

A uterus corresponding in size to the period of pregnancy; a varying amount of hæmorrhage and pain, with a corresponding degree of collapse; widening of the cervix at the cervico-vaginal junction; and a slightly patulous condition of the external os, are suggestive of threatened abortion.

Marked ballooning of the cervical canal; closure of the os externum, which has a thin parchment-like edge; and a varying amount of dark, perhaps putrid discharge, are indicative of cervical abortion.

The expulsion of a portion of the ovum; slight diminution in size of the uterus; a patulous cervical canal; and a profuse dark discharge, or even a sharp hæmorrhage, are indicative of incomplete abortion.

The expulsion of the entire ovum; a trumpet-shaped condition of the cervix; a marked diminution in size of the uterus; and the gradual diminution and final disappearance of all discharge, are indicative of complete abortion.

A cessation of development, or a gradual diminution in size of the uterus; a dark hæmorrhagic discharge, which may be fœtid; disappearance of the subjective and objective symptoms of pregnancy, and general ill-health on the part of the patient, are suggestive of missed abortion.

The existence of an ovoid pulsating tumour at one side of the uterus; a history of pregnancy; an enlarged uterus, corresponding in size to, or slightly smaller than, the date of pregnancy accounts for; slight irregular hæmorrhages; and perhaps the expulsion of a decidua in which no trace of chorionic villi or amnion can be found, are suggestive of an unruptured extra-uterine pregnancy.

A varying degree of hæmorrhage; the expulsion of a decidua in which no trace of chorion or amnion can be found; considerable pain; collapse out of proportion to the amount of external hæmorrhage; and perhaps the disappearance of a previously discovered pelvic tumour, with the formation of a new tumour in Douglas' pouch, are suggestive of a ruptured extra-uterine pregnancy.

Marked increase in size of the uterus, out of proportion to the period of pregnancy; a profuse watery blood-stained discharge, perhaps containing cysts; and crampy pains, are indicative of myxoma chorii.

B. HÆMORRHAGES OCCURRING DURING THE SECOND THREE MONTHS

Two varieties of hæmorrhage are met with during the second three months of pregnancy:—

I. Hæmorrhage due to detachment of the placenta.

II. Hæmorrhage due to extra-uterine pregnancy.

I. Hæmorrhage due to Detachment of the Placenta

Hæmorrhages due to detachment of the placenta, occurring during the second three months of pregnancy, occupy an intermediate position between abortion and accidental and unavoidable hæmorrhages.

With the exception of a small proportion of cases which are due to degeneration of the ovum, we find that, while pathologically they can be classified in the same manner as can the hæmorrhages of the last four months, clinically they must be treated as those of the first three. The reason of this is obvious. In the four last months of pregnancy it is always possible to distinguish clinically between the two great classes of placental insertion; in the second three months of pregnancy it is not possible to do so, although pathologically the two classes occur. Consequently, while in the four last months we can diagnose exactly the nature of the case, and treat it accordingly in the most suitable manner, in the second three months all we can do is to recognise that the patient is bleeding, and that the hæmorrhage is either so trifling as only to require palliative treatment, or so serious as to demand more radical measures.

Etiology.—As has been pointed out above, the hæmorrhage may come from a placenta which is normal in position, or from one which is prævia. The distinction between the two, which cannot be made prior to the expulsion of the ovum, can be made in most cases subsequently. This is done by noting the relationship of the opening in the membranes, through which the fœtus was expelled, to the placenta, if there is such an opening. If the opening is quite close to the placenta, or even through it, it shows that the placenta must have been implanted quite close to, or right over, the os. In some of these cases, however, when the ovum is expelled spontaneously, it is unruptured; while in those cases in which it has to be removed, the membranes become so torn that it is impossible to determine the above relationship. Even in the cases in which the placenta is certainly prævia, it is doubtful whether the situation is the cause of its detachment, or whether rather, the latter is not really due to such similar causes as act in the case of a normally seated placenta,—the fact that

the placenta is prævia being a chance accompaniment. It is not very easy to explain why a placenta prævia should be detached any more readily than a normally situated placenta before the lower uterine segment has commenced to form, *i.e.* before the ninth month. The causes of the detachment of a normally situated placenta are to be found in some diseased condition of itself or of the decidua serotina. Accordingly, the following may be stated to be the causes of placental detachment :—

(*a*) Endometritis, by causing an unhealthy condition of the decidua.

(*b*) White infarction of the placenta due to Bright's disease.

(*c*) Syphilis, by affecting either the placenta or decidua. And perhaps

(*d*) Insertion of the whole or part of the placenta in the lower uterine segment.

Symptoms.—The essential symptom of the case is the occurrence of hæmorrhage, perhaps accompanied by the death of the fœtus. In the latter case, decomposition may result, if the membranes are ruptured; or, on the other hand, if they remain intact, the fœtus may merely macerate or mummify aseptically. In the large majority of cases, the hæmorrhage is external, *i.e.* it escapes from the uterine cavity as rapidly as it finds its way into the latter from the blood-vessels. In a smaller proportion of cases, on the other hand, it may remain stored in the uterus, which gradually increases in size to suit the demands of the accumulating blood. If the area of placental detachment is so great as to leave an insufficient amount of surface in contact with the uterus to provide the necessary nutrition and oxygen for the fœtus, the latter dies. In most cases, this event will determine the onset of labour, but in some cases a condition analogous to missed abortion results, and the ovum is retained *in utero*. On the other hand, if there is still a sufficient area of placenta intact to keep the fœtus alive, the latter continues to grow. The condition of the uterus will depend on the foregoing. If the fœtus lives and the hæmorrhage is external, the uterus will continue to increase in size in proportion to the advance of pregnancy. If the fœtus dies, the hæmorrhage being still external, the uterus will diminish in size as the liquor amnii is absorbed, and there will be in addition the symptoms due to the retention of a dead fœtus. The breasts become flaccid, and any secretion which may have appeared in them disappears. At the same time, the subjective symptoms of pregnancy lessen, and the patient suffers to a varying degree from the absorption of ptomaines from the dead fœtus. If the hæmorrhage is internal and the fœtus dead, the patient suffers in the same manner from the retention of the fœtus, but the uterus increases in size. In such cases there will be great difficulty in feeling the fœtal parts; and the uterus, which ordinarily in hæmorrhage is softer than usual, will, if the bleeding is excessive, become extremely tense and hard. Finally, if the membranes have ruptured and saprophytic germs have found their way into the cavity, the fœtus will decompose and a fœtid discharge result.

Accordingly, we may sum up the symptoms as follows :—A varying amount of hæmorrhage, either external or internal. A gradual increase in the size of the uterus, if the fœtus lives and the hæmorrhage is external. A diminution in size, if the fœtus dies and the bleeding is external. A more or less marked and sudden increase, if the hæmorrhage is internal. A putrid discharge, if the fœtus decomposes.

Diagnosis.—The diagnosis of external hæmorrhage is obvious. The diagnosis of internal hæmorrhage is made by noting the increase in size of the uterus—an increase which enlarges the uterus out of proportion to the period of pregnancy—by the difficulty in feeling the fœtal parts, and by the general condition of the patient. Internal hæmorrhage at this stage of pregnancy is most likely to be confused with myxoma chorii, but here a mistake in diagnosis is not of any great importance, as in either case the uterus must be emptied. The nature of the discharge will in most cases enable a distinction to be made, as in hæmorrhage it is grumous and contains clots; in myxoma chorii it is thin and watery, while, if myxomatous cysts are found, the nature of the case is at once apparent.

Treatment.—Our treatment of these cases is palliative or active, according to their nature. If the hæmorrhage is slight, and there is no evidence that the fœtus is dead, every effort must be made to check the bleeding by any means short of emptying the uterus. The main factor in attaining this end is absolute rest in bed for at least ten days or a fortnight after hæmorrhage has ceased. There are certain drugs which, according to some authorities, can produce a tonic effect upon the uterus or its blood-vessels sufficient to check slight hæmorrhage. Of these, the chief are ergot and strychnine administered in conjunction. Atthill recommends the continued administration of a mixture containing 15 minims of Ext. Ergot. Liq. and 5 minims of Liq. Strychninæ. Hydrastis Canadensis has also its advocates. The objections which are so constantly urged against ergot do not hold good in the case of the last-mentioned drug. Ergot produces its effect by causing a tonic contraction of the uterine muscle fibres, and in all probability only produces such effect when the contractions have already started of their own accord. Accordingly, in the case of a patient who is bleeding, but who is not in labour, ergot will probably produce little or no effect; while, if

any contractions of the uterus have occurred, they will be increased by the ergot given, and so any possibility of their passing off and allowing the pregnancy to continue will be removed. Hydrastis Canadensis, on the other hand, is said to produce its effect by direct action on the muscle fibres of the blood-vessels, and so does not tend to accelerate the occurrence of uterine contractions. The usual dose of it is from fifteen to twenty minims.

Active treatment, consisting in the emptying of the uterus, is indicated under the following conditions:—

(1) If it is obvious that the patient has lost as much blood as is safe.

(2) If the discharge is fœtid.

(3) If the fœtus is dead.

If the indication for delivery is hæmorrhage alone, and there is no sign of intra-uterine decomposition, the easiest method of emptying the uterus consists in passing as many sea-tangle tents into the cervix as the latter will hold, after carefully disinfecting the external genitals and vagina, and then in plugging the vagina tightly with iodoform gauze. It is better to use a number of small tents in preference to a couple of large ones, as the dilating effect of the former is superior. The plug and tents are to be removed in twenty-four hours, or sooner if labour pains ensue. If there is no appearance of the latter when the tents are removed, it will be well to rupture the membranes, to draw down a foot into the vagina, and either to leave the further expulsion to the uterine contractions, or to complete the delivery of the fœtus. In all cases, if contractions do not follow the rupture of the membranes within four to six hours, it will be best to empty the uterus, as there is always a danger of decomposition occurring if we wait too long. An alternative treatment to the use of sea-tangle tents is the plugging of the utero-vaginal canal with iodoform gauze after the method of Dührssen. To do this, the cervix is drawn down by means of an American forceps on both lips, after thorough disinfection of the vagina, and is dilated with Hegar's dilators as far as possible without laceration. The end of a long strip of iodoform gauze, two inches wide, and either single or double as is preferred, is passed by means of a sound as far into the uterus as possible. This is followed by the remainder of the strip, and when it is finished, by another similar strip which is knotted on to the first. Each bit of gauze is passed as high into the uterus as possible, and so the maximum amount introduced. As soon as the cavity is filled, the vagina is plugged tightly. This plug is left *in situ* for twenty-four hours, or until strong labour pains ensue, and is then removed. The advantage of this method is that all hæmorrhage is checked, that intra-uterine decomposition is prevented, and that, even if labour does not follow its removal, there is then sufficient dilatation of the cervix to permit of the easy extraction of the fœtus.

In those cases in which hæmorrhage is complicated by the occurrence of decomposition, the emptying of the uterus must be carried out at one sitting. This is always a more or less troublesome operation owing to the small size of the cervix, and frequently it will only be possible to extract the child after some proceeding akin to embryotomy. The author has found the following plan to be rapid and comparatively easy. Disinfect the vagina thoroughly, and dilate the cervix as far as possible with Hegar's dilators. Pass one finger into the uterus, seize one, or if possible both feet, and draw them down as far as possible, which will usually be until the breech comes into the cervical canal. As soon as they cannot be drawn down any farther, introduce a Schultze's spoon-forceps, or other forceps of a similar shape, into the uterus alongside the breech, and catch the latter in it and pull it down. This will result in the tearing off of part of it, and then a fresh hold is taken, and so on until the body of the child is extracted piecemeal. The head will be found the most difficult to remove, as it has the largest diameter, but by means of the forceps it can be crushed and so extracted. A Schultze's spoon-forceps is a very safe instrument for such a course of procedure, as, owing to its shape, it has no tendency to catch the uterine walls, while it easily seizes anything which is lying in the cavity. As soon as the fœtus has been entirely removed, the finger is again passed into the uterus and the placenta detached, while counter-pressure is made upon the fundus by the hand placed on the abdominal wall. The uterus is then well douched with a solution of creolin and water (1 in 320), or other antiseptic, and the cavity plugged with iodoform gauze. The latter must be removed in from twelve to twenty-four hours, and the uterus again douched if there is any rise of temperature.

Prognosis.—The prognosis in these cases is good unless the patient has been allowed to become very much weakened by constant hæmorrhage, or unless she has absorbed an excessive dose of ptomaines from the decomposed fœtus. For this reason the uterus must always be emptied as soon as any of the indications for active treatment, as given above, show themselves.

II. Hæmorrhage due to Extra-Uterine Pregnancy

When the ovum escapes into the broad ligament, and survives that event, the condition is known as a mesometric pregnancy. The peritoneum which forms the broad ligament now occupies the relationship to the ovum which the tube formerly did, and as a result it is liable to share the same fate, *i.e.* secondary rupture of the gestation sac may occur. The immediate

result of this rupture depends upon the position of the placenta. If the placenta is situated above the fœtus, it will almost certainly be involved by the rupture; and if this occurs during the latter half of pregnancy, the hæmorrhage will be so profuse as to cause the death of the patient (Bland Sutton). If the placenta lies beneath the fœtus, the latter can escape into the abdominal cavity, when rupture occurs, without necessarily causing very profuse hæmorrhage. The symptoms and treatment of this condition, which are practically the same as those of primary rupture, will be found in the article "Ectopic Gestation" (vol. iii. p. 13). When rupture occurs it may be accompanied by slight uterine hæmorrhage, and perhaps by the expulsion of a decidua.

C. HÆMORRHAGES OCCURRING DURING THE LAST FOUR MONTHS

Hæmorrhages occurring during the last four months may be due to:—

I. Detachment of the placenta.
II. Rupture of the uterus.

I. Hæmorrhage due to Detachment of the Placenta

Cases of hæmorrhage from detachment of the placenta are divided into two great classes:—

A. Accidental hæmorrhage, due to the detachment of a normally situated placenta.

B. Unavoidable hæmorrhage, due to the detachment of a placenta prævia.

A. Accidental Hæmorrhage

Accidental hæmorrhage is the term applied to hæmorrhage due to the detachment of a normally seated placenta, *i.e.* a placenta no part of which comes into the lower uterine segment.

Frequency.—The frequency of accidental hæmorrhage is very difficult to ascertain, on account of the cursory manner in which the subject is treated in the majority of text-books. At the Rotunda Hospital, amongst 15,109 cases of labour there were 113 cases of accidental hæmorrhage, *i.e.* one in every 133·7 cases. Almost all these cases were cases of external accidental hæmorrhage.

Etiology.—The most frequent cause of detachment of the placenta is to be found in some factor which weakens the adhesions between the latter and the uterine wall. Of these, the commonest are endometritis, Bright's disease, and syphilis. Of the three, Bright's disease is the most important, and is practically the sole cause of the more serious forms of accidental hæmorrhage. It favours the rupture of vessels in the placental substance, and blood escaping from them forces its way between the placenta and the uterine wall, thus causing the detachment of the former. Another cause of quite a different nature from the foregoing is to be found in undue shortness of the cord, either actual or the result of the twisting of the latter round the fœtal parts. When such a condition is present, so great traction may result upon the placenta while the fœtus is being expelled as to cause detachment.

Varieties.—Two chief forms of accidental hæmorrhage are met with, according as the blood escapes externally, or is stored up in the uterus. These are:—

(A) Concealed accidental hæmorrhage.
(B) External accidental hæmorrhage.

These two varieties differ so markedly from one another, as far as the conditions present and the appropriate treatment are concerned, that they must be described separately.

(A) *Concealed Accidental Hæmorrhage.*—Concealed accidental hæmorrhage is the term applied to accidental hæmorrhage when the blood is stored up in the uterus instead of escaping into the vagina. In such a condition the blood may be found in one of four situations:—

(1) Behind the placenta, the whole of which is separated except its edges.

(2) Behind the membranes, which are detached except round the internal os.

(3) In the amniotic cavity.

(4) Behind the presenting part, if the latter fills the lower uterine segment completely.

Concealed accidental hæmorrhage is, with the exception of acute sepsis, the most serious accident which can happen to a pregnant woman. This is due not only to the difficulty of diagnosing its occurrence at an early stage, but also to the fact that we have an entirely different condition of the uterus from that which we find in external hæmorrhage. In concealed hæmorrhage the blood collects in the uterus, because the latter dilates so easily that the intra-uterine pressure is never sufficiently great to overcome the slight resistance offered to its outflow through the cervix. In external hæmorrhage quite the opposite is the case. Here, the intra-uterine pressure is so rapidly raised, by the escape of blood from behind the placenta, that such blood is almost immediately forced through the cervix. The cause of the difference in the two cases is, that in internal hæmorrhage we have to deal with a uterus the muscle fibre of which has for some reason lost its normal contractile tone, and whose elasticity is, as a result, impaired; while in external hæmorrhage, the uterine fibre possesses that normal tone, and hence may be described as "healthy." It is quite obvious that a ruptured vessel can only bleed into a closed cavity as long as the pressure inside that cavity is less than the blood pressure. Once they equalise one another the bleeding ceases. If the blood can escape from the cervix as quickly as it flows from behind the placenta, then such equalisation never takes place. If the uterus is healthy in tone and the outflow of blood from the cervix is prevented, the two

pressures rapidly equalise one another and the bleeding ceases. If, however, the uterine fibre has lost its tone to such an extent that it will permit the uterus to dilate before the blood pressure, then not only does the intra-uterine pressure never rise sufficiently to check the hæmorrhage, but it is not even sufficient to force the blood which has escaped through the cervix. In this manner concealed hæmorrhage commences. The amount of hæmorrhage which the uterus will permit to accumulate inside will depend upon the extent to which the muscle fibre is affected. The first sign that the latter is commencing to react against the distension it is undergoing, is the escape of blood externally, and accordingly the earlier that occurs the healthier may the condition of the uterus be judged to be. These are all important facts to grasp, as they explain the essential differences between external and internal hæmorrhages so far as accompanying conditions go. They also indicate a means by which external hæmorrhage may be checked, and the necessity for uterine co-operation in carrying out such a treatment.

Symptoms. — The symptoms of concealed hæmorrhage fall under two heads:—

(1) Those due to the loss of blood, and common to all forms of hæmorrhage.

(2) Those due to the accumulation of blood in the uterus.

(1) The first group does not require any special attention here. The symptoms are given in full in the section dealing with post-hæmorrhagic collapse (*v.* "Labour, Post-partum Hæmorrhage," vol. v. p. 256).

(2) The most prominent symptom in the second group consists in the gradual enlargement of the uterus, which may dilate to such an extent as to fill the entire abdomen and cause pressure on the diaphragm. The uterus at first becomes tense, and then of an almost woody hardness, and is markedly tender to the touch. All possibility of palpating the fœtus disappears. At the same time there is severe abdominal pain, akin to the pain caused by the tonic contraction of the uterus in cases of threatened rupture.

Diagnosis.—The diagnosis of concealed hæmorrhage due to detachment of the placenta can be made by means of the symptoms given above.

Tenderness of, and increase of size in, the uterus, with ever-increasing pain, are almost pathognomonic of intra-uterine hæmorrhage—an important fact to bear in mind even when there is a sufficient amount of external hæmorrhage to account for any other symptoms.

Treatment. — The treatment of concealed hæmorrhage is so far by no means satisfactory. Mild cases which occur during labour are not very difficult to treat, but severe cases starting prior to the onset of labour are most serious. From what has gone before, it will be obvious that plugging of the vagina—a line of treatment which is so satisfactory in external hæmorrhage—is here of no avail. In those cases in which we are able to recognise the very commencement of the hæmorrhage, and while still the patient shows no marked symptoms of bleeding, some good may be gained by the application of a tight abdominal binder, and possibly by the administration of ergot; while the application of a firm vaginal compress—as presently described—may bring on labour before the hæmorrhage has reached a formidable amount. Once the patient begins to have strong uterine contractions a great deal of the risk of the case disappears. Immediate rupture of the membranes is then indicated, with the object of permitting the blood to escape and the uterus to contract down upon the fœtus. The remaining treatment of the case is the same as that of external accidental hæmorrhage when the patient is in labour. Unfortunately, many cases of concealed hæmorrhage have a sudden onset before the patient is in labour, and do not admit of any temporising measures. In such cases the bleeding must be immediately checked, and in order to do this the seat of the hæmorrhage must be directly reached. Two lines of treatment present themselves—*accouchement forcé*, and supra-vaginal amputation of the uterus either by Porro's method or not, as may be preferred. These are both most serious measures, and convey *per se* a great element of danger into the case. The only excuse for their adoption is that the danger which they are directed against is so urgent that even the risk of Porro's operation, or *accouchement forcé*, with a collapsed patient, is to be preferred.

By *accouchment forcé* is meant the dilatation of the cervix followed by version and the extraction of the fœtus. It is performed as follows:—Place the patient in the cross-bed position, with her hips well over the edge of the bed; if possible an anæsthetic should be given. The first step is the dilatation of the cervix. This can be best accomplished, in cases in which the inner os is dilated, by means of several incisions through the walls of the cervical canal, radiating outwards from the os externum. This, however, can only be practised in primiparæ, in whom the os internum is found dilated previous to the onset of labour. In multiparæ, where the cervix is still long and the inner os closed, mechanical dilatation must be practised. This is usually performed by means of the fingers. First one finger is introduced gently through the cervix, and then, equally gently, another beside the first. Then a third finger, and so on, until finally the whole hand can be introduced. If there is not sufficient dilatation at the start to allow of the introduction of one finger, a few Hegar's dilators must be used, and by this means the cervical canal enlarged. As soon as the whole hand has been passed

into the uterus, the foot of the fœtus is seized and drawn down. By gradually pulling upon it the size of the cervical canal is still further increased, and finally the breech can be brought through. The remainder of the fœtus is then extracted in the usual manner. If the placenta is not immediately expelled by the uterine contractions, it should be removed by the hand, and in all cases the utero-vaginal canal must be plugged firmly with iodoform gauze.

Supra-vaginal amputation of the uterus is perhaps to be preferred to the foregoing. It is easier if there is sufficient assistance. It is quicker if Porro's operation is performed. There is less blood lost during its performance, and there is no more, if as much, shock. The objection to it is the greater number of assistants which it requires. As to the method of performing it there is nothing very special to be said. For the description of the operation the reader is referred to the article on "HYSTERECTOMY," vol. v. p. 304. Porro's operation is the most rapid method, but convalescence is slower than if an intra- or retro-peritoneal treatment of the stump is adopted.

Prognosis.—The prognosis for the mother is very serious, especially in those cases which come on before labour starts; for the child it is almost absolutely bad.

(B) *EXTERNAL ACCIDENTAL HÆMORRHAGE.*—External accidental hæmorrhage is the term applied to accidental hæmorrhage when the blood escapes from the uterus according as it is poured out from behind the detached placenta. It is a serious accident, but by no means as serious as is concealed hæmorrhage. In the majority of cases the blood pours into the vagina as rapidly as it escapes from the ruptured vessels. Sometimes, however, a certain amount of blood may first accumulate in the uterus, and then commence to escape externally as soon as the latter reacts against the blood pressure, *i.e.* as soon as the intra-uterine pressure becomes sufficiently great to overcome the resistance to the escape of blood.

Symptoms.—The escape of blood from the vagina is the most marked symptom, accompanied by the usual symptoms of collapse if the bleeding continues long enough. In those cases in which there is concealed hæmorrhage as well, the symptoms which have been given under the heading of concealed hæmorrhage are met with.

Diagnosis.—The diagnosis of the case has to be made from hæmorrhage due to placenta prævia, *i.e.* unavoidable hæmorrhage. To do this, examine the patient vaginally, with the object of determining whether the placenta can be felt in the lower uterine segment or not. If it can be felt, it is a case of placenta prævia; if it cannot, of accidental hæmorrhage. In some cases in which the placenta cannot be felt, it may possibly be a case of lateral placenta prævia, but still it is to be treated as if it was one of accidental hæmorrhage. The reason for this is that, if the placenta lies at a height at which it cannot be felt from the vagina, though actually it may be a placenta prævia, still the treatment suitable for accidental hæmorrhage will be found to be the more efficacious.

Accidental hæmorrhage may be diagnosed by abdominal palpation by excluding the possibility of placenta prævia. If the head is found to be fixed in the pelvis, and the patient is bleeding, it is certainly not a case of placenta prævia.

Treatment.—The treatment of the case, as well as the prognosis, depends to a very great extent upon the time at which the hæmorrhage starts, *i.e.* whether during pregnancy or labour—when there are no uterine contractions, or when there are. If the patient is in labour, the danger of the case is greatly diminished, and the treatment is comparatively simple. If she is not in labour, the reverse is the case.

If the patient is not in labour when the hæmorrhage starts, there are two points towards which our treatment must be directed. The first is the immediate checking of the hæmorrhage; the second, the induction of labour. If the hæmorrhage can be satisfactorily stopped for the time necessary to bring on uterine contractions, the case is practically reduced to one of hæmorrhage during labour, and the prognosis consequently improved. As has been shown above, there is very little room for blood to accumulate in a healthy uterus filled by an unruptured ovum. Either it must escape from the cavity as rapidly as it pours out from behind the placenta, or the intra-uterine pressure will become equal to the blood pressure, and the bleeding cease. Again, the fact that the hæmorrhage is external tends to show that the uterus is "healthy," as is demonstrated by the statistics of cases treated on this basis. In no case of accidental hæmorrhage at the Rotunda Hospital has plugging converted an external into an internal hæmorrhage, an accident which would surely occur if the uterine fibre had lost its tone. If we prevent the blood which is being poured out from behind the placenta from leaving the uterus, the pressure inside the latter will rapidly become equal to the blood pressure, and the hæmorrhage will cease. The easiest and most effectual method of doing this is by plugging the vagina tightly, and so compressing the cervix. This will check the hæmorrhage, and at the same time it will carry out our other object, which is a necessary part of any successful treatment, namely, it will induce labour. Furthermore, labour thus brought on comes on gradually, and does not cause any aggravation of the shock from which the patient is already suffering, as do the more rapid methods of emptying the uterus. On the contrary, ample time is allowed for the

patient to rally from the collapse, which the hæmorrhage has caused, before the uterus empties itself.

The operation of plugging is carried out as follows:—Place the patient in the cross-bed position, with her hips well over the edge of the bed. Administer an anæsthetic if necessary, and this is most frequently the case, as the firm application of a plug which is required in these cases is rather a painful procedure. Wash the external genitals and douche out the vagina thoroughly. Then pass a posterior speculum, and, with strips of iodoform gauze soaked in lysol solution (0·5 per cent), plug tightly round and over the cervix. A speculum is not absolutely necessary, but, if the patient is not under an anæsthetic, it renders the proceeding less painful by protecting the perineum. The remainder of the vagina is then plugged as tightly as possible with balls of cotton-wool about the size of a golf-ball, which have been

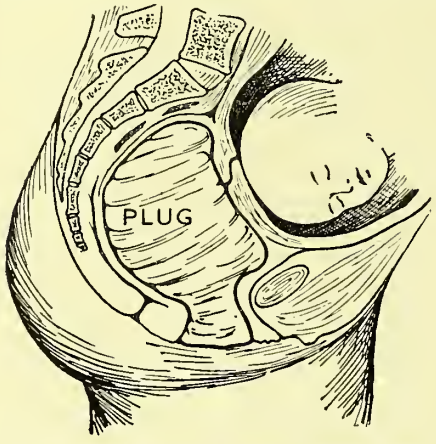


FIG. 2.—Diagram showing a vaginal tampon *in situ*.

previously sterilised by boiling and then soaked in lysol solution. This plugging is continued until no more can be pressed into the vagina (*v.* Fig. 2). The patient is then put back to bed, and an abdominal binder pinned as tightly as possible round the uterus, while a T-binder is brought down between the thighs, and also fixed firmly. By this means the uterus is compressed between the plug in the vagina and the abdominal binder, and the intra-uterine pressure is raised. The plug is left in until strong labour pains ensue, which usually happens in from two to four hours. In some cases the onset of labour is slower than this, and in such the plug must be removed in twenty-four hours for fear of decomposition taking place. If hæmorrhage comes on again it can be replaced, but this is rarely necessary. While the plug is *in situ* the patient must be carefully watched to see that concealed hæmorrhage is not occurring. The first sign of such a condition is afforded by the patient complaining that the abdominal binder is becoming tighter than she can bear—a condition which is caused by the increase in size of the uterus.

The success of this treatment depends upon three points:—

(1) The uterus must be "healthy."
(2) The ovum must be intact.
(3) The plug must be tightly applied.

If the patient is in labour when the hæmorrhage starts, the first thing to be done is to rupture the membranes. This treatment, which is frequently recommended as *the* treatment in all cases, is now most suitable. Every time the uterus contracts it drives the presenting part or the liquor amnii against the membranes lying over the os internum, and these latter in turn communicate this downward impulse to the placenta. In this manner, with each contraction, while the membranes are unruptured, a fresh piece of placenta is detached. When the membranes are ruptured the presenting part can advance without causing any such drag upon the placenta, and at the same time, owing to the escape of liquor amnii, the uterine cavity is diminished in size, thus causing a corresponding diminution in the placental site. Rupture of the membranes can be accomplished by means of a stilette or the finger-nail. Care must be taken to ensure the gradual escape of the liquor amnii, especially in those cases in which the presenting part is not fixed, lest the cord should be carried down at the same time. In addition to rupturing the membranes, a hot vaginal douche may be given to stimulate uterine contractions, and massage of the fundus, with the same object, may also be tried.

If such treatment fails to check the bleeding, our further plan of action depends upon the size of the os. If the latter is still quite small, the vagina must be plugged as before. This treatment, which would be most unsafe after the membranes are ruptured, if there were no labour pains, becomes again safe if there are strong contractions, as the latter ensure the obliteration of any space round the fœtus, left by the escape of the liquor amnii. The plug is left *in situ* until pains of an expulsive character set in. It may then be removed. The use of the plug in cases such as this is, however, very seldom required, as in the large majority of cases hæmorrhage ceases when the membranes are ruptured in the presence of uterine contractions, or else it is possible to empty the uterus. If the os is half or more dilated, and the presenting part is not fixed, podalic version, followed by extraction, is the line of treatment indicated. If, however, the patient is very much collapsed, and all hæmorrhage ceases as soon as a foot has been brought down into the cervical canal, it may be advisable to leave the expulsion of the child to the natural efforts. Version in these cases can usually be carried out by the internal method; in some cases it may be necessary to adopt the bi-polar method, as will be presently described.

If, on the other hand, the os is fully or almost fully dilated and the head is fixed, immediate delivery by the forceps is indicated.

Other Modes of Treatment.—The above is the treatment which the writer recommends, his reason for which will be found below. But, in addition, there are other methods which are recommended by various authorities. Of these the following are the chief:—

Rupture of the membranes in every case.—Rupture of the membranes in every case is the treatment which has perhaps the greatest number of supporters. It is a treatment which is easily carried out, and in some cases is sufficient. If we can be certain that the uterus will contract down upon the fœtus immediately after the escape of the liquor amnii, there are no very great objections to trying such a course of action, as if it fails it is still possible to resort to plugging. But, if the uterus does not contract down upon the fœtus, there is a large space left inside it for blood to collect in, consequently plugging is impossible; and, if hæmorrhage continues, there is nothing left but *accouchement forcé*—the most dangerous of all treatments. Accordingly, rupture of the membranes should be left for those cases in which we can be certain that, after it is done, the uterus will contract down; that is, it should only be performed when the patient is in labour.

Accouchement forcé.—*Accouchement forcé*, as has been stated before, is the most dangerous of all treatments. It is quite unjustifiable in any case of external hæmorrhage so long as the membranes are intact. The great objection to it lies in the fact that intra-uterine manipulations, particularly the violent manipulations of *accouchement forcé*, tend directly to aggravate the collapse into which the patient falls in cases of severe hæmorrhage. Delivery, even when effected by the natural efforts, increases the collapse, and consequently any treatment which will check the hæmorrhage, and also afford an interval during which the patient can revive before the uterus is emptied, is the treatment that is to be preferred.

Prognosis.—The prognosis in external accidental hæmorrhage depends very much on the form of treatment adopted. In the Rotunda Hospital, 56 cases of accidental hæmorrhage of all degrees of severity were treated between November 1889 and November 1893. *Accouchement forcé* was the mode of treatment adopted in all the serious cases, and of those so treated 6 died. From November 1893 to November 1900, 57 cases were treated. *Accouchement forcé* never was performed, its place being entirely taken by plugging. Out of this number 1 patient died, and she had been admitted to the hospital thirty minutes previously with the membranes ruptured. The number of serious cases during these two periods was relatively the same. The treatment by rupture of the membranes in all cases is credited with a mortality of 10 to 12 per cent by its supporters; and yet a well-known obstetrician used to consider that the fact of a student recommending plugging of the vagina as a treatment for accidental hæmorrhage was sufficient ground for depriving him of his examination. The subject is so important that the writer wishes to state again that the advantages of the vaginal plug lie in the following facts:—That it at the same time checks the hæmorrhage and brings on labour; that labour so induced comes on gradually, and at a time when the patient has had an interval of rest during which she can recover from her collapsed condition; that the vaginal plug applied in suitable cases does not tend "to convert an external into an internal hæmorrhage"; and that, even if the bleeding continues after the plug has been applied, the patient is in no worse position for the adoption of any other treatment.

The fœtal mortality is very high—from 40 to 60 per cent. It will be discussed subsequently

B. Unavoidable Hæmorrhage

Unavoidable hæmorrhage, or hæmorrhage due to placenta prævia, is the term applied to the bleeding caused by the detachment of a placenta, any portion of which is implanted so near the os internum, that it becomes separated during the formation of the lower uterine segment.

Frequency.—The statistics relating to the frequency of placenta prævia are very conflicting. Kaltenbach gives the proportion of 1 in 1500 to 1600, Winckel 1 in 1500, Ribemont-Dessaignes 1 in 1000, the Boston Lying-in Hospital 1 in 239. At the Rotunda Hospital, among 15,109 cases of labour there were 86 cases of placenta prævia, or 1 in 175·68.

Etiology.—The etiology of placenta prævia is very obscure. It is more frequent amongst multiparæ than amongst primiparæ, and is also relatively more frequent in the case of twin pregnancies. Of the many theories which have been brought forward, three are specially worth attention.

(1) That, owing to the effects of a previous endometritis, the cavity of the uterus has become so enlarged that its walls are no longer in contact with one another. Consequently, when an ovum enters the uterus through the Fallopian tube, instead of being detained at the fundus by the pressure of the mucous membrane, it drops downwards and becomes attached to the lower portion of the uterus. Support is given to this theory by the fact that placenta prævia almost always occurs in patients who have suffered from endometritis, as shown by menstrual disturbances, leucorrhœa, and abortions.

(2) Hofmeier and Kaltenbach attribute placenta prævia to a portion of the placenta being developed out of chorionic villi which are implanted in the decidua reflexa, instead of only out of those that are implanted in the decidua serotina. The tongue of placenta thus formed becomes attached to the lower pole of the uterus as the ovum grows.

(3) That a placenta prævia is merely a normally situated placenta, which, owing to its size, has extended into the lower uterine segment. This may be the case in some instances, but in the majority of cases the placenta is no larger than is normal.

Whatever may be the cause of the low insertion of the placenta, the bleeding which results from it is started by the expansion of the lower pole of the uterus consequent upon the formation of the lower uterine segment during the later months of pregnancy. As a result, the placental site becomes too large for the placenta, and detachment ensues (Jacquemier). Pinard thinks, on the other hand, that the detachment is due to the painless contractions of the uterus, which, by diminishing the size of the middle and upper portions of the cavity, tend to increase the size of the lower, and so to pull it away from the placenta.

Occasionally hæmorrhage may occur, due, not to the detachment of the placenta, but to the rupture of the circular sinus, a large vein which runs round the placenta. This is an accident which is specially likely to occur when the edge of the placenta is over the os, but its occurrence has also been noticed in cases of normally situated placentæ.

Varieties.—A placenta prævia is termed central, marginal, or lateral, according as it completely covers the os, just reaches its edge, or merely extends into the lower uterine segment without reaching the inner os (*v.* Fig. 3). It will be at once obvious that these relationships will materially alter according to the size of the os. Thus, what may be apparently a central placenta prævia at the commencement of labour, will as the os dilates become marginal, and so it is essential for clearness' sake to specify the size of the os at the time at which the examination is made.

Symptoms.—The chief symptom is, the occurrence of an attack of hæmorrhage, coming on without any apparent cause, and of more or less severity. The first attack of hæmorrhage, if not treated, may be sufficiently profuse to cause the death of the patient, but such an occurrence is very rare. More commonly she recovers from it, only to have a second attack in from eight to ten days, and this in turn is followed by others. The hæmorrhage of placenta prævia is, essentially, a hæmorrhage of repetitions (Ribemont-Dessaignes). As in accidental hæmorrhage, the bleeding is most severe during a contraction of the uterus. This, in placenta prævia, is due to the fact that the vessels which supply the placenta lie below the contraction ring, and consequently during a contraction not only are they not obstructed, but the pressure in them is increased, owing to the obstruction offered to branches running above the contraction ring. If the bleeding continues, the usual symptoms of collapse appear.

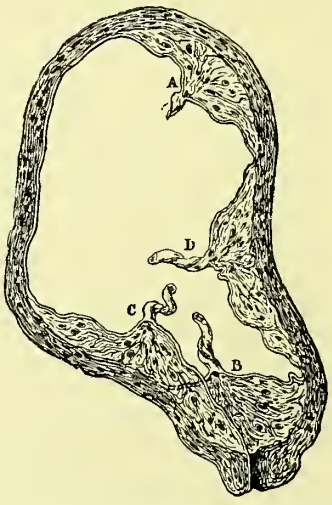


FIG. 3.—Diagram representing the various situations of the placenta. A, Normal situation; B, placenta prævia centralis; C, placenta prævia marginalis; D, placenta prævia lateralis. (Modified from *The Norris Text-book of Obstetrics.*)

Diagnosis.—There is but one really reliable method of diagnosing a placenta prævia, that is by feeling it through the cervical canal, or, if that is closed, through the lateral fornices. In almost all cases in which our attention is called to the condition by hæmorrhage, the cervix will be found to be sufficiently dilated to admit two fingers, so that there is rarely any difficulty in determining the position of the placenta. The latter is felt as a spongy mass, either completely covering over the os internum or lying to one or other side of it (*v.* Fig. 4). A blood-clot which has become adherent to the membranes in either of these situations is the thing most likely to be confounded with it. A distinction can be made by noting the manner in which a clot can easily be squeezed through between the fingers, while a portion of placenta cannot. If the cervical canal is closed, the placenta can be felt as a thick soft mass lying between the uterine wall and the presenting part at one or other side. The cervix and the lower uterine segment are also softer than usual, and the vessels of the lateral fornix are enlarged and pulsate more strongly than normal. If the placenta cannot be felt, the case is to be treated as one of accidental hæmorrhage.

A placenta prævia may also be sometimes diagnosed by abdominal palpation. This can be done by noticing that there is something which either displaces the presenting part to one or other side of the false pelvis, or prevents it from descending through the brim. Then, on very careful palpation, a soft mass may be felt at the side from which the presenting part has been displaced. If the presenting part has become fixed in the brim, it is almost

certain that the case is not one of placenta prævia.

Treatment.—The first point to recognise with regard to the treatment of placenta prævia is that, immediately the condition is recognised, steps must be taken with the view of ending the pregnancy, whether the bleeding has temporarily ceased or not. The only exception which may be made to this rule, is in those

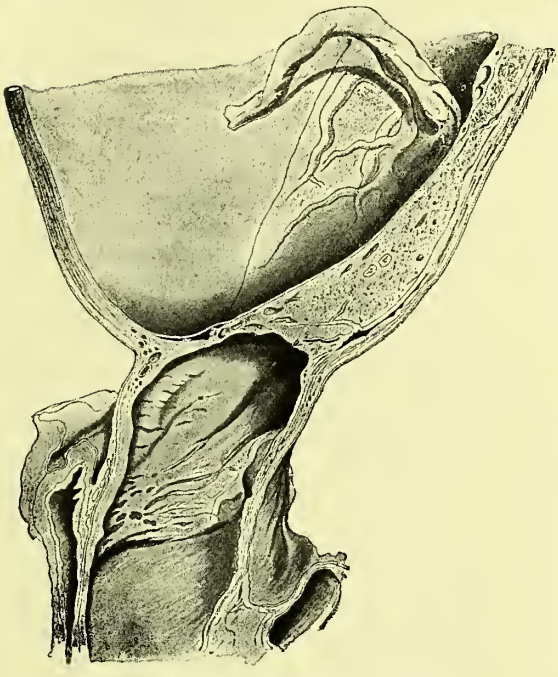


FIG. 4.—Marginal placenta prævia. (Ahlfeld.)

cases in which the patient is in such circumstances that, if the bleeding restarts, she can be immediately treated. Many cases have been lost because, on the arrival of the doctor, the bleeding was found to have ceased, and consequently nothing was done, with the result that the bleeding recurred, with fatal consequences.

As in accidental hæmorrhage, the line of treatment adopted depends to a very great extent upon whether the patient is in labour or not. If she is in labour, the treatment of the case is comparatively simple, both because uterine contractions are present, and also because the fact that hæmorrhage did not occur until the patient came into labour, shows that the placenta is not very close to the os.

Again, as in accidental hæmorrhage, the objects of our treatment are to check the bleeding and to bring on labour gradually. These objects are best attained by the method introduced by Braxton Hicks. This consists in turning the child by podalic version into a breech presentation, rupturing the membranes, and drawing down a foot. The remainder of the expulsion of the fœtus is then left to nature. The result of this treatment is that the hæmorrhage is checked, owing to the pressure of the breech and subsequently of the body of the child against the placenta, while the rupture of the membranes and the partial extraction of the fœtus ensure the onset of labour. If there is any return of the bleeding, a little traction on the foot will drag more of the breech downwards, and so increase the pressure upon the placenta. If, as may happen in very rare cases, labour pains do not come on within twelve hours, or if, even before that time—the fœtus being dead—signs of decomposition set in, delivery must be accomplished by means of gentle traction upon the foot. The method of performing bi-polar version, which the author prefers, differs somewhat from the more classical method of Braxton Hicks, and so a short description of it may not be out of place. The operation is carried out as follows:—Place the patient in the cross-bed position, and under an anæsthetic, unless her condition forbids the use of one. Ascertain the exact position of the fœtus by abdominal palpation, and then by external version turn the fœtus so that it lies directly transversely—its head at one side of the uterus, its breech at the opposite, and its back uppermost. By this means the legs of the child are brought to lie in the region of the os internum. This is the first part of the operation. The second part is commenced by washing the external genitals thoroughly and douching out the vagina. Then as much of the hand as is necessary is passed into the vagina, and two fingers introduced into the cervix. If the placenta is central, the fingers must be passed through it, and the foot subsequently brought down through the opening thus made. By firm external pressure on the breech with the other hand, the feet are made to descend still farther, and then one of them can be seized between the fingers in the uterus. As the cervical canal is usually very small, the foot must be drawn down in such a manner as to take up the least room, *i.e.* by bringing the toes first and the rest of the foot in an extended position. In some cases it may happen that the os, which is large enough to admit two fingers by themselves, or a foot by itself, will not allow the three to come down together. In such a case the following manœuvre is sometimes successful. With the fingers passed through the cervix, draw the tips of the toes downwards through the inner os as far as possible. Next remove the fingers and keep the foot in the same position by means of external pressure upon the breech. Then, with the fingers in the vagina, encircle the cervix and push it up gently over the toes, while at the same time the external hand presses the breech down. By this means half of the foot is brought outside the os externum, and is caught by the fingers. A little traction then

draws it down (*v.* Fig. 5). At the same time that this final traction is being made, the abdominal hand pushes the fœtal head upwards to the fundus of the uterus, and so version is completed. The last step consists in tying a strip of iodoform gauze to the foot, as by this means traction can be most easily applied if necessary.

In order that this line of treatment can be carried out satisfactorily two conditions must be fulfilled:—

(1) The cervical canal must be of sufficient size to admit two fingers at least. If the hæmorrhage is at all severe this condition is practically always fulfilled.

(2) The membranes must be intact, or only quite recently ruptured. If the uterus has

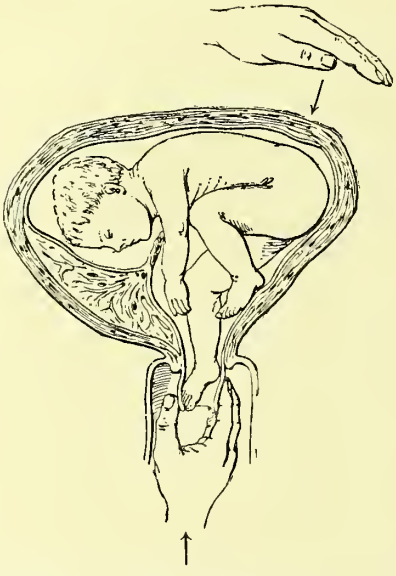


FIG. 5.—Method of completing bi-polar version in a case in which the size of the os will not permit of the presence of the foot and the two fingers at the same time. The hand in the vagina pushes the cervix upwards while the foot is made to descend by pressure upon the breech (diagrammatic).

become contracted down upon the fœtus it will be found impossible to turn except by internal version, and there is rarely or never sufficient cervical dilatation to allow of this. This condition will also be always fulfilled unless an ignorant attendant has resorted to rupture of the membranes, with the idea that such treatment is sufficient.

In the rare case in which the cervical canal is not sufficiently dilated to admit two fingers, and in which the hæmorrhage is so severe that it is impossible to wait for a short time to allow it to dilate, the firm plugging of the vagina with iodoform gauze and cotton-wool is indicated. This is performed in the same manner as has been already described under the head of accidental hæmorrhage.

When the patient is in labour, the treatment of the case is the same as that of accidental hæmorrhage occurring under the same conditions. Commence by rupturing the membranes, a proceeding which is usually sufficient. This acts by allowing the head to advance without causing traction through the membranes upon the placenta, and at the same time the descending head presses upon the placenta and checks hæmorrhage, as does the breech after version has been performed. If this is not sufficient to check the bleeding, internal version may be performed, followed or not by extraction, according to the condition in which the patient is and the size of the os. If the head is fixed and the os sufficiently dilated, the forceps may be applied.

Other Modes of Treatment.—As in accidental hæmorrhage, the above is the treatment which the author considers most suitable. Other lines of treatment have been recommended by various authorities, and of these the following are the chief:—

(1) Champetier de Ribes' Bag.—The plugging of the lower uterine segment by means of a Champetier de Ribes' bag is perhaps the line of treatment which, after bi-polar version, has the greatest number of advocates at the present day. A Champetier de Ribes' bag is a pear-shaped bag made of waterproofed silk. Its wide end or base is three-and-a-half inches across, while the narrow end tapers to the size of a quarter-inch rubber tube, with which it is continuous, and by which it is filled. The bag, or hydrostatic dilator as it is termed, is used as follows:—The patient is placed in the cross-bed position, with her hips well over the side of the bed, and the vagina is thoroughly disinfected. The membranes are then ruptured immediately over the inner os, and the bag is passed upwards into the amniotic cavity by means of a forceps made for the purpose. It is then distended slowly by injecting into it any antiseptic fluid, either by attaching a syringe, the tube of a uterine douche, or other form of irrigator, to the nozzle. As soon as it is full the tap is turned off to prevent the fluid escaping. Finally, the end of the bag or colpeurynter is connected with the foot of the bed by means of an elastic cord, which is made as tight as the patient can bear. The effect of the introduction of a pear-shaped bag like Champetier de Ribes' into the amniotic cavity is to cause a compression of the placenta against the uterine wall, and at the same time a dilatation of the cervical canal, in very much the same manner as the breech of the child does in Braxton Hicks' method; while the rupture of the membranes also helps to bring on uterine contractions. The bag is allowed to remain in the uterus as long as it will, that is, until the cervical canal is so dilated that it slips out. If this does not happen within twelve hours the bag must be removed, the child turned by bi-polar or internal version, according to the

size of the os, and extracted immediately or not, according to the condition of the patient. The average time which the bag takes to dilate the cervix is said to be five hours and ten minutes (Blacker). In the case of a central placenta prævia, the bag must be introduced through an opening in the placenta made over the inner os.

The advantages claimed for this course of procedure over Braxton Hicks' method are the ease with which it can be carried out, and the improved fœtal prognosis. While fully recognising it as a perfectly scientific mode of treatment, and one which gives excellent results, the writer considers it to be inferior, for general use, to version. In careless hands it is more liable to cause sepsis, inasmuch as a possibly non-sterile foreign body is lying for some hours in the uterus; and its performance, which may not be required very frequently, necessitates the use of a special apparatus, and one, moreover, which is very liable to be destroyed by lying by.

(2) *Accouchement forcé.* — The adoption of *accouchement forcé* is even more dangerous in the case of placenta prævia than it is in the case of accidental hæmorrhage, owing to the condition of the cervix. It is most essential to remember that in these cases no attempt may be made to deliver the fœtus through an incompletely dilated os.

(3) Plugging of the Vagina.—Plugging of the vagina is an objectionable and unnecessary course of procedure in placenta prævia. There is always some risk of sepsis following the use of a plug, and this risk is very much increased in those cases in which the placenta is situated in the lower uterine segment, as the inoculation of the placental site by the extension of vaginal infection is so easy. Further, we have an excellent and safe mode of treating these cases which we do not possess in the case of accidental hæmorrhage, viz. version. Accordingly, the use of the plug should be limited to the small proportion of cases in which it is absolutely necessary, viz., to those cases in which the patient is bleeding, and in which the cervical canal is not sufficiently dilated to admit even two fingers.

(4) Partial Detachment of the Placenta.—Detachment of the placenta from the lower uterine segment was the main feature of the treatment recommended by Barnes. The steps of his procedure, in his own words, are as follows:—

"(*a*) Rupture the membranes; this disposes the uterus to contract.

"(*b*) Apply a firm bandage over the abdomen.

"(*c*) A tampon may be introduced to gain time, but it is not necessary to do so. Watch; observe with vigilance.

"(*d*) Detach all the placenta adhering within the inferior zone, and always watch. If there is no hæmorrhage, wait a little. The uterus may perhaps do what is necessary. If this fails, dilate the cervix with the hydrostatic dilator. Wait and watch. If the natural forces fail, employ the forceps, which gives the best chance to the child, or as a last resort perform version.

"(*e*) Avoid as far as possible everything that predisposes to septicæmia."

Barnes' treatment has now but very few supporters. At the time at which it was introduced it was a considerable advance over the methods in use, inasmuch as it prevented the rapid emptying of the uterus. It is, however, very much inferior to either Braxton Hicks' method or to the use of Champetier de Ribes' bag. It is a very tedious course of procedure for the patient, and one which affords many opportunities of inoculating her with septic organisms. In placenta prævia, more than in any other condition, the number of vaginal examinations must be brought down as near the irreducible minimum as possible, on account of the ease of infecting the placental site. But Barnes' method demands repeated examinations in order to carry out its multiple steps, and to ensure that the necessary "vigilance" is being used.

Complications.—Although the main danger caused by placenta prævia is death from hæmorrhage coming from the placental site, there are still other grave risks sufficiently imminent to require careful consideration during the treatment of a case. Of these the following are the chief:—

(1) Laceration of the Cervix.—As has been already mentioned, laceration of the cervix is very prone to occur in these cases, owing to the softened condition of the cervix resulting from its increased blood supply. Further, for the same reason that they occur, they are the more dangerous, as, if any of the large vessels going to the placenta are torn across, fatal traumatic hæmorrhage will very probably result. It is this which makes *accouchement forcé* so dangerous a proceeding in these cases, and which furnishes a direct contra-indication to the extraction of the child through an incompletely dilated cervix. If post-partum hæmorrhage follows the birth of the child in these cases, the possibility of its being due to a laceration must always be borne in mind.

(2) Septic Infection.—In consequence of the proximity of the placental site to the vagina, the absorption of septic organisms, or of ptomaines, may very easily take place. Such absorption may occur not only during labour, but also during the peurperium. Accordingly, as has already been said, vaginal examinations must be avoided as far as possible during labour, and any appearance of sapræmic change in the vaginal contents after confinement must be immediately treated.

(3) Post-partum Hæmorrhage.—Post-partum hæmorrhage in cases of placenta prævia is a comparatively common occurrence. Its frequency is due to three factors:—

(*a*) Laceration of the Cervix.—This has been already noted.

(*b*) The Situation of the Placental Site.—When a large portion of the placenta extends below the contraction ring, its site cannot be affected to the same extent by the subsequent contraction and retraction of the uterine muscle as if the placenta was entirely situated in the contractile part of the uterus. As a result, the lumen of the placental vessels may not be completely obliterated.

(*c*) The Debilitating effect of previous Hæmorrhage upon the Patient.—Marked anæmia and debility, the results of previous hæmorrhages, have frequently a very prejudicial effect upon the uterine contractions.

Prognosis.—The prognosis in placenta prævia depends greatly upon the treatment adopted. The maternal mortality is said to vary between 2 and 40 per cent. Galabin had 92 cases, with 8 deaths; Winckel, 19 cases, with 1 death; Dührssen, 50 cases, with 2 deaths. Blacker has collected 22 cases in which Champetier de Ribes' bag was used, with 1 death. At the Rotunda Hospital, where Braxton Hicks' method is used in all severe cases, 86 cases were treated from 1st November 1889 to 1st November 1900, with 3 deaths. Of the 3 deaths, one was due to rupture of the uterus occurring in a case in which the forceps were applied. A second was due to hæmorrhage, the patient being admitted moribund, after a journey of five miles in an open cart. The third died of sapræmia, from which she was suffering when admitted. As in accidental hæmorrhage, the fœtal mortality is very high—from 40 to 60 per cent.

Fœtal Mortality in Accidental and Unavoidable Hæmorrhage.—A few words must be said with regard to the fœtal mortality in these cases. It has been found by experience that the life of the child is, in antepartum hæmorrhage, more or less antagonistic to the life of the mother, and that any treatment which will give the lowest maternal mortality will give the highest fœtal, and *vice versa*. *Accouchement forcé*, which in the past gave a maternal mortality of from 40 to 60 per cent, gave a considerably lower fœtal mortality than does the treatment which is adopted at the present day. So far no treatment has been described which, while affording the same excellent maternal results that are obtained by Braxton Hicks' treatment in placenta prævia, or by vaginal plugging in accidental hæmorrhage, materially reduces the fœtal mortality. This condition of affairs is after all no more than must be expected. So far as we can see at present, it is necessary to give the mother as much time as possible to recover from the hæmorrhage which has occurred, before the uterus is emptied either naturally or artificially. During this time, the fœtus is limited in its supply of oxygen to the amount which can come through a placenta, of which not only a large portion is detached, but of which the remainder is undergoing a more or less forcible compression against the uterine wall. If this compression can obliterate the vessels which are torn across, it must also to some extent diminish the size of the lumen of those which are intact. Furthermore, even if the fœtus is delivered alive, its expectation of life is extremely bad. It is frequently premature, and has always suffered more or less from deprivation of oxygen. As a result, such infants very frequently die during the first month after birth. Under these circumstances, the author considers that attempts should not be made to save the life of the child, if such attempts will materially affect the prognosis for the mother. It may frequently seem, in placenta prævia, as if a slight traction upon the leg of the child would save its life by hastening its delivery, and the very natural desire to save both lives may induce us to do so. In some cases, where the condition of the mother is good and the cervix is sufficiently dilated to remove the danger of laceration, it may be possible to hasten delivery without increasing the maternal danger to an unjustifiable extent. On the other hand, if the case is one which should have been left to nature—as the majority are—we may only find that we have sacrificed the life of the mother for the sake of an infant who succumbs shortly after its birth.

II. Hæmorrhage due to Rupture of the Uterus

Hæmorrhage always accompanies rupture of the uterus. Its amount varies according to the site of the laceration, the involvement or escape of the large uterine vessels, the amount of pressure exerted by the fœtal parts upon the laceration, the strength with which the uterus subsequently contracts, and the extent to which the neighbouring parts are implicated. The hæmorrhage may be internal, *i.e.* into the peritoneal cavity, or external, according to the site of the laceration, according as it is complete or incomplete, and according as the situation and nature of the presenting part prevents or permits the escape of blood. The symptoms are those of internal or external hæmorrhage, as the case may be, with the addition of the more direct symptoms due to the laceration itself (*vide* the articles on "Rupture of the Uterus," Labour, "Injuries during," vol. v. p. 264).

2. Arising independently of the Pregnancy

MENSTRUATION

The possibility of menstruation occurring during pregnancy is a question which apparently

has not been definitely decided. In answering such a question much depends upon the definition we attach to the word menstruation. If the latter is understood to mean merely a periodical discharge, there is apparently no reason why it should not occur. Undoubtedly the uterus undergoes some form of stimulation, even during pregnancy, at what would have been the menstrual periods if the patient had not been pregnant. Under such circumstances, it is not very difficult to believe that the attendant congestion may cause a slight hæmorrhagic discharge from an ulcerated cervix, a polypus, or even from the inflamed decidua vera covering the lower pole of the uterus before this membrane has come into contact with the decidua reflexa, that is, before the third month. If, however—as is the more correct definition—we consider the term to be applicable only to the physiological discharge which accompanies the monthly destruction and expulsion of the uterine endometrium, it is obviously impossible that menstruation could take place during pregnancy without causing abortion. The cases of supposed menstruation during pregnancy can almost invariably be accounted for by some pathological condition of the uterus or cervix. The regular occurrence of a monthly discharge during the whole of pregnancy points almost invariably to the existence of a double uterus. Pajot says that, while the hæmorrhagic discharges which occur during pregnancy may have some characters which cause them to resemble menstruation, they invariably differ from the latter in their duration, and in the quantity and quality of the blood. Pinard states that there has never yet been observed a single case of the persistence of menstruation in a pregnant woman. Dakin considers that, "while one monthly bleeding may be allowed to pass as a menstruation in the absence of any discoverable cause, or of any further disturbance, any repetition of this should always be looked upon as a threatening of abortion, and the patient treated on this assumption."

American authorities, on the other hand, do not regard the persistence of menstruation as so impossible or improbable. Lusk considers that the occurrence of pregnancy is not incompatible with the existence of a periodical flow—to which, however, he is careful not to apply the term menstruation. Parvin states that a monthly flow may occur once or oftener during pregnancy, or even persist during the whole period. While Palmer, of Ohio, records the case of a patient of his own who never menstruated except when she was pregnant!

The general attitude of modern authorities, in the case of patients who consider themselves to be pregnant while they are still menstruating, may be summed up in the words of Stoltz: "Rarely will one be deceived who regards a woman menstruating regularly, with all the characters of menstruation, as not pregnant; while trusting the contrary opinion, he is exposed to frequent errors."

HÆMORRHAGE FROM TUMOURS

Myomata.—Myomata of the uterus generally cause sterility, and consequently are not a very frequent complication of pregnancy. Intrauterine myomata may, during pregnancy or labour, give rise to accidental hæmorrhage by causing the detachment of the placenta, owing to the irregularity their presence imparts to the uterine contractions. Subsequent to delivery they are a very common cause of post-partum hæmorrhage. Myomata of the cervix, or pedunculated myomata of the uterus which project into the vagina, may give rise to constant small hæmorrhages during pregnancy, and have been found as the cause of those periodical discharges which simulated menstruation.

Treatment.—For the treatment of myomata of the body of the uterus complicating pregnancy, the reader is referred to the article on these tumours. Small or large polypoid myomata projecting into the vagina or cervical canal must be twisted away, or cut away, according as is necessary. The constant hæmorrhagic discharge which they cause weakens the patient; there is a great risk of their being deprived of their blood supply and sloughing, and they may offer an obstruction to the delivery of the child when labour comes on. There is little or no risk of their removal causing the onset of labour.

Malignant Disease.—Malignant disease of the cervix or vagina will cause a more or less continuous hæmorrhagic and perhaps sanious discharge during the whole of pregnancy. It will also favour the occurrence of abortion owing to the anæmia and cachexia it induces. The rapidity of its growth is very marked during pregnancy, while after the expulsion of the fœtus there is a temporary check.

Treatment.—If the existence of malignant disease is determined in the early months of pregnancy, abortion ought to be induced, whether the case is one in which a radical operation is possible, or whether it is not. In the former, the induction of labour is a matter of course; in the latter, it should be performed in order to check the otherwise rapid growth. If the patient is in the later months of pregnancy, and the radical removal of the growth is possible, labour ought to be induced irrespective of the condition of the fœtus. If the growth is inoperable, and our decision lies between the extraction of a premature or immature fœtus through the vagina, or a full-term fœtus by Cæsarean section, we must be largely or entirely influenced by the wishes of the patient. In a case in which we determine to wait, a temporary check may be given to the discharge by curetting away any very prominent masses

which project into the vagina, and by careful cauterisation.

Other tumours or pathological conditions, whose existence may give rise to slight hæmorrhage coming from the vagina or its neigbourhood during pregnancy, are urethral caruncle, mucous polypi of the cervix, and hæmorrhoids.

HÆMORRHAGE FROM TRAUMATISMS

Traumatisms in the region of the vagina and vulva will cause a varying degree of hæmorrhage, according to the nature of the lesion. Their treatment is similarly influenced. Considerably more interesting than the immediate effect of such injuries is the question of the result of a traumatism, either surgical or otherwise, upon the pregnancy. So far, the most varying consequences have been met with. In some women, a very severe accident or surgical operation has been attended by no bad effects upon the pregnancy. In other women, the slightest accident or interference has been followed by the immediate emptying of the uterus. Guéniot's conclusions on this subject are as follows:—

(1) The harmlessness of traumatism in pregnancy is not governed by any absolute law.

(2) A traumatism, if the woman is without morbid predisposition—she, her uterus, and the ovum healthy—is generally without injurious effect upon the pregnancy.

(3) If gestation is complicated by a pathological condition, such as abnormal irritability of the uterus, disease or great size of the ovum, albuminuria, etc., the traumatism, however slight, and whatever the part involved, most frequently causes premature expulsion of the ovum.

(4) Great caution is necessary in performing surgical operations during pregnancy.

Ribemont-Dessaignes considers that accidental traumata vary in their effects, according to their intensity, their site, the amount of hæmorrhage which accompanies them, and the occurrence of wound complication, sepsis, etc. As regards surgical intervention Verneuil says: "Surgical intervention is not forbidden during pregnancy, but is under the guidance of a definite rule—to abstain from it when it is possible to do so; to intervene when it is necessary."

Prehallux.—The hypothetical accessory digit on the foot, which is believed to have been suppressed during evolution.

Premature Infants. *See* Couveuse; Labour in Multiple Pregnancy (*Infantile Risks, Prematurity*); Labour, Faults in the Passenger (*Hydramnios*); Labour, Obstetric Operations (*Induction of Premature Labour*); Pregnancy, Pathology (*Intra-uterine Diseases*).

Premature Labour. *See* Labour, Obstetric Operations (*Induction of Premature Labour*).

Premaxilla.—The anterior fourth of the palatal process of the superior maxilla is known as the premaxilla; it is ossified from the incisive centre; and it has been thought to consist of two parts (endognathion and mesognathion) carrying the central and the lateral incisor tooth respectively.

Premenstrual.—Occurring before menstruation, *e.g. premenstrual pain.* *See* Menstruation and its Disorders (*Dysmenorrhœa, Cause of the Pain, Congestion*).

Premolar. *See* Teeth (*Genesis of the Teeth, Permanent*).

Premonitory. — Forewarning or indicating beforehand, *e.g.* the premonitory signs of labour or of a disease.

Prenatal.—Occurring before birth; antenatal. *See* Antenatal.

Preparations.—In Anatomy preparations are parts of the body so preserved as to be useful for teaching purposes; in Pharmacy they are the forms in which drugs are compounded, *e.g.* standardised preparations (those containing a certain fixed proportion of the principle of the drug).

Prepollex.—A suppressed digit supposed to have existed on the radial side of the thumb (pollex). *See* Prehallux.

Prepotency.—The fact that in heredity and in respect to certain characters the paternal inheritance is often more potent or more capable of finding expression in the progeny than the maternal, or *vice versa.* *See* Heredity (*Degrees of Hereditary Resemblance*).

Prepuce. *See* Herpes (*Herpes Genitalis*); Circumcision; Penis, Surgical Affections of (*Preputial Calculi*); Phymosis and *Cross References.*

Presbycusis. — Senile deafness (Gr. πρέσβυς, an old man; ἀκουή, hearing).

Presbyopia.—Senile defect of vision (Gr. πρέσβυς, an old man; ὤψ, the eye). *See* Accommodation (*Presbyopia*); Headache (*Diagnosis of the Cause, Age*); Refraction (*Presbyopia*).

Prescribing, and Methods of Administering Drugs.

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PRESCRIBING

GENERAL PRINCIPLES. — Many years ago Dr. Paris summarised the aim of prescribing—that is, the art of combining medicinal substances —as :—(1) To augment, correct, or modify the action of a drug. ; (2) to obtain the joint action of two or more diverse remedies ; (3) to form a new remedy ; (4) to obtain a suitable form. Applications of these principles readily suggest themselves ; thus combinations of purgatives each of which operates in a different way are more effective than single drugs, while in the same manner chloral and opium together act as a hypnotic with less disturbance to other functions than is produced by a sufficient dose of either given separately. Again, a cough mixture may contain morphia to allay the cough, and ipecacuanha to facilitate expectoration, while combinations containing ferrous sulphate and an alkaline carbonate form a new remedy—ferrous carbonate.

There is no doubt that skill in the art of writing prescriptions is cultivated to a less extent now than in a former generation : for this there are several reasons. A more scientific therapeutics has led to the use of simpler formulæ, and prescriptions containing only one or two drugs are much more common than in the past. Polypharmacy has declined, and with it some of the rather mystical importance attached to an "elegant" prescription. At the same time, the constant influx of new synthetic remedies, and the exploiting of what should be the domain of the physician by the manufacturing chemist in the production of combinations of drugs in forms more attractive and convenient than the six-ounce mixture, have still further contributed to inducing practitioners to order quasi-proprietary remedies (and excellent many of these preparations are) in preference to thinking out an appropriate prescription for the case in hand.

No one will deny that the modern pharmacy is a great advance upon the old, and instead of railing at the general use of drugs in tablet and other forms by the public, the duty of the medical man is to correct his own ways, and to adapt to his own prescriptions the newer methods of the pharmacist, as a very little forethought will enable him to do.

WEIGHTS AND MEASURES.—The weights and measures in common use in this country for prescriptions are the grain (gr.), the drachm (ʒj = 60 grs.), and the ounce (℥j = 8 drs.). The scruple (℈j = 20 grs.) is practically obsolete. For fluid measure we have the minim (♏j), the fluid drachm (fl. ʒj = 60 min.), and the fluid ounce (fl. ℥j = 8 drs). These are troy or apothecaries' weight ; in the Pharmacopœia only the avoirdupois weight is recognised, which differs from the preceding in having 437½ grs. to the ounce, and 16 ozs. to the pound, instead of 480 grs. and 12 ozs. respectively. In prescribing, however, the apothecaries' or troy system is used. Advisable though the decimal system may be for other purposes, it has not as yet, in this country, made much headway as a means of writing prescriptions. One practical drawback to its use is that, while to reap the full advantage of a decimal prescription the mixture must be made up in ten or a multiple of ten doses, yet since we order the doses to be taken at stated intervals of time, which is reckoned on a duodecimal system, it is generally more convenient to give three, four, six, or twelve doses, to be taken at corresponding hourly intervals, than to give five or ten. On the other hand, it is very often convenient to order drugs for external application as percentages, in which case we may either adopt the metric system of weights, or prescribe so many parts per cent of each ingredient, and direct the dispenser to dispense an ounce or more of the combination. Tables for the conversion of the decimal to other systems, and *vice versa*, are so readily accessible that it is unnecessary to give them here. It should, however, be remembered that a gramme is just about equal to 15 grains (15·432) and a cubic centimetre to 17 minims (16·9), while a milligramme equals $\frac{1}{64}$ grain and a centigramme about $\frac{1}{6}$ grain.

As domestic measures the tablespoon, dessertspoon, and teaspoon are commonly treated as the equivalents of half an ounce, two drachms, and one drachm respectively. The use of a correctly graduated measuring glass is, however, advisable. An ordinary tablespoon contains about six drachms, a dessertspoon three drachms, and a teaspoon one and a half drachms ; an afternoon teaspoon holds somewhat less than a drachm. A liqueur glass holds practically an ounce, and a sherry glass about two ounces and a half. These measures are fairly constant. The size of a drop varies greatly according to the viscosity of the fluid ; it is a very rough measure of a minim. Thus while water gives 50 or 60 drops to the drachm, alcohol, tinctures, and fluid extracts give about 140, and chloroform gives 250. It may at least be said, therefore, that when drops are counted as minims there will be no likelihood of overdosage.

Types of Prescriptions.—The technique of prescription writing may best be illustrated by taking a typical example, such as the well-known cough mixture of the Brompton Hospital. In its unabbreviated form it runs :—

Superscription	Recipe.	
Inscription .	Liquoris Morphinæ Hydrochloridi	drachmas duas (*Basis*).
	Acidi Hydrocyanici Diluti	drachmam unam (*Adjuvant*).
	Syrupi Tolutani	unciam unam (*Corrective*).
	Infusum Rosæ Acidum	ad uncias sex (*Vehicle*).
Subscription .	Misce.	

Signature.—Signa (or Signetur)—One tablespoonful to be taken in water every four hours.

Initials of Prescriber.

Patient's Name and Address.

Date.

The above illustrates the classical component parts of a prescription. We have first the *Superscription*—Recipe ; next, the *Inscription* or body of the prescription, with its *Basis* (the morphia), its *Adjuvant* (the dilute hydrocyanic acid), its *Corrective* (the syrup of tolu), and its *Vehicle* (the infusion of roses). Then comes the *Subscription* or direction to the dispenser—Misce ; and finally, the *Signature* or instructions to the patient. The objects of the four ingredients are sufficiently indicated by the words basis, adjuvant, corrective, and vehicle ; and although not all prescriptions contain these, the traditional plan of combining four substances is still very frequently followed.

Grammar of a Prescription.—The Latin required for a prescription is extremely simple, and the tendency is more and more to limit it. The above illustrates some of the chief points to be attended to. Recipe is the second person singular of the imperative mood of the verb *recipio*, and means "take thou." The contraction ℞ by which it is replaced in everyday work is the symbol of Jupiter—the last relic of the mediæval physician-astrologer's invocation of planetary help and guidance. The names of the first three drugs are in the genitive—of the liquor of the hydrochloride of morphine, etc.—and the quantities of each in the accusative, after the active transitive verb *recipio*. The vehicle is generally written in the accusative and followed by *ad* when not an exact quantity, but only the amount requisite to bring the whole mixture up to a given bulk is stated. The convenience of this is obvious, since when solids are ordered by weight their bulk is not so precisely known as to make it possible to say how much fluid must be added to yield (say) six ounces. All drugs, however, of which the required amount is exactly stated, should be put in the genitive, the word *ad* being omitted.

In the usual contracted form the prescription would run thus :—

January 18, 1906.

℞ Liq. Morph. hydrochlor.	℥ij.	
Acid. hydrocyan. dil.	℥j.	
Syr. Tolu.	℥j.	
Inf. Rosæ acid. ad	℥vi.	M.

Sig.—One tablespoonful in water every four hours.

A. B.

Mrs. Smith,
10 High Street.

The directions to the patient are seldom now written in Latin, but, as we have said, the custom of using that language for the body of the prescription still obtains. This is perfectly defensible on two grounds: first, it prevents the patient from knowing exactly what he is getting—often a very desirable matter ; second, it enables a pharmacist unacquainted with English to dispense the prescription. It is rarely necessary now to give minute directions as to pharmaceutical procedures, hence the Latin of the subscription is greatly simplified. Practically all the phrases in common use are given in the table on page 301 (Table I.).

The following formulæ (which are given uncontracted) will serve to exemplify further what has been said, and to indicate the most common modes of expressing prescriptions :—

Recipe, Tincturæ digitalis.		
	Tincturæ nucis vomicæ, ana	drachmam unam.
	Spiritus ammoniæ aromatici	drachmas sex.
	Aquæ	uncias duas.

Fiat Mistura.

Signa.—A dessertspoonful in water every four hours.

Recipe, Santonini		grana quatuor.
	Hydrargyri subchloridi .	grana sex.

Misce et divide in pulveres quatuor.

Signa.—One powder to be taken at bed time.

Recipe, Podophylli resinæ . .		Semi granum.
	Extracti hyoscyami . .	Granum unum cum semisse.

Fiat pilula secundum artem. Mitte tales duodecim.

Signa.—One to be taken after dinner.

In contracted form the last would run :—

℞ Podophyll. res. gr. ʃs.
Ext. hyoscyam. gr. i/s.
F. pil. sec. art. Mitte tal. xii.
Sig.—One to be taken after dinner.

One or two other practical points call for notice. Prescriptions should be clear. This is only attainable by legible writing and the avoidance of such ambiguous contractions as Ac. hyd. dil., etc., which may mean either hydrochloric or hydrocyanic acid ; Chlor. hyd., which may mean chloral hydrate, calomel, or corrosive sublimate, and the like.

When the physician does not wish a prescription to be repeated without his sanction—*e.g.* in the case of morphia, cocaine, etc.—it is usual to add to the subscription the words *Non rep.*, for "non repetatur." In the case of

poor persons, to whom the cost of medicine may be a serious consideration, the insertion of the letter "P" in the subscription may be used to indicate this; most druggists dispense prescriptions so marked at the lowest possible price, but it should be remembered that this is an act of grace, and that the courtesy ought never to be abused. The name and address of the patient should always be written on the prescription.

The *directions to the patient* ought to be written in full, invariably stating *how* (in water, etc.) and *when* (at what intervals, or in what relation to food) the doses are to be taken. The hasty "as directed" is too often a cloak for negligence, and when it is used the prescriber ought to be sure that the patient really has received such instructions as may enable him to use the medicine as was intended. Still, when ordering remedies for use in menstrual disorders, urethral discharges, etc., it is obviously undesirable to write explicit directions on the prescription: here, "as directed" finds a proper sphere.

In ordering a medicine it is usual to state whether it is to be taken before or after food, and in doing so we are guided by the effect which we wish to produce. If a local effect on the stomach is desired, or if we wish the drug to act rapidly, it should be given on an empty stomach. On the other hand, drugs which are in any way likely to irritate the stomach or disturb digestion should be given after a meal, so that they may be diluted by the gastric contents. In the case of drugs given for the express purpose of influencing digestion, the time at which they are taken depends on the circumstances of each case. Thus alkalis given to correct acidity are administered at a short interval after a meal; hydrochloric acid, to remedy defective gastric secretion, during meals; saline aperients, again, should be taken in the morning, while fasting.

Drugs given in small bulk and soluble form are more rapidly absorbed than when insoluble or much diluted.

Table I

Contraction.	Latin.	English.
āā	ana	of each.
ad		to, up to.
ante cib.	ante cibos	before food.
Aq.	Aqua	water.
aq. dest.	aqua destillata	distilled water.
bis die	bis in die	twice daily.
caps.	capsula	capsule.
collyr.	collyrium	an eye-wash.
c̄.	cum	with.
div. in part. æq.	divide in partes æquales	divide into equal parts.
ft.	fiat, fiant	let it (them) be made.
garg.	gargarisma	a gargle.
haust.	haustus	a draught.
M.	misce	mix.
mass.	massa	a pill mass.
mitte		send.
mit. tales	mitte tales	send of such.
non rep.	non repetatur	not to be repeated.
pt. æq.	partes æquales	equal parts.
post cib.	post cibos	after food.
p. r. n.	pro re nata	as needed.
q. s.	quantum sufficit	as much as is sufficient.
rep.	repete	repeat.
sec. art.	secundum artem	according to art.
semi-(gran.)	semi-(granum)	half-(a-grain).
ʃs.	(granum unum cum) semisse	(one grain and) a half.
Sig.	Signa, Signetur	write, let it be marked.
S.	solve	dissolve.
t. i. d.	ter in die	thrice daily.

Dosage.—The two principal factors influencing dosage are the *Age*, or size, of the patient, and *Susceptibility*.

Age.—Strictly speaking, it is the bulk of the patient, rather than his age, which should affect the dose; but in practice not much account is usually taken of this, further than that on the whole a somewhat smaller dose of a drug is generally given to a woman than to a man. In the case of children the usual custom is to give a fraction of the adult dose equal to $\frac{x}{x+12}$, where x = age. Thus if a drachm be the adult dose of a remedy, to a child of four we should order $\frac{4}{4+12}$, or one fourth of a drachm. Other methods of reckoning the dosage for children have been suggested, but the above, which is applicable to most drugs (the chief exceptions are noted below), is as simple and satisfactory as any. In old age smaller quantities of most drugs (except aperients) are required than in the young adult, but no definite rules can be laid down.

Susceptibility.—This is influenced chiefly by age, sex, certain physiological conditions, and personal idiosyncrasy.

Age. — Children, as is well known, are peculiarly intolerant of certain remedies, yet bear relatively large doses of others. In the former category the chief place must be given to opium, which ought to be administered to infants and young children with great caution. At one year ½ to 1 minim of laudanum is an average dose; compound tincture of camphor may be given during the first year to the extent of one minim for each month, while at

one month about $\frac{1}{20}$ minim of laudanum may be administered. Digitalis, quinine, alcohol, strychnine, and mercury are well borne by children; while purgatives, chloral, antipyrin, arsenic, and belladonna are tolerated in doses relatively much larger than by adults. In the aged, who so often suffer from bronchial catarrh, thickened vessels, and inadequate kidneys, opium must be employed with caution. Tonics are seldom well borne, in particular those containing strychnine. This drug should indeed be given in very small doses to old people, whether as a stimulant in acute illness, or as a tonic in debility.

Idiosyncrasy.—The chief drugs towards which patients may be expected to show personal idiosyncrasy are antipyrin and antifebrin, quinine, bromides and iodides, cocaine, belladonna, arsenic, iron, and copaiva.

Such physiological states as *pregnancy* and *lactation* are reputed to be influenced by certain drugs, hence care should be taken in prescribing during those periods. In pregnancy, drugs such as ergot, quinine, drastic purgatives, and lead ought to be given cautiously for fear of producing abortion, while during lactation saline aperients and belladonna, which may interfere with the secretion of milk, ought to be avoided. Certain drugs, too, are excreted by the mammary glands, and may thus affect the infant. The importance of this has probably been greatly overrated. Thiemich (*Monatschrift f. Geburtshülfe u. Gynaecologie*, 1899, Bd. x.), in a critical review of the whole subject, shows that the only drugs which assuredly pass over in the milk are salicylates and iodides. In one instance it seemed as though mercury also did so. Briensa (*Ztsch. f. Exp. Path. und Therapie*, 1907, Bd. iv. Hft. 2), after administering medicinal doses of a large number of drugs to nursing women, could detect only iodides, bromides, iron, arsenic, aceto-salicylic acid, and calomel (as mercury) in the milk. Experimentally, traces of opium, atropine, and (when given in toxic doses) alcohol have been detected in the milk, but so far as the therapeutic administration of drugs to nursing women is concerned the only ones in which this question need be considered are salicylates and iodides, rhubarb and senna, which may cause catharsis in the infant, and thyroid extract, which has produced symptoms of thyroidism in the breast-fed infants of mothers taking the drug.

Toleration.—After long-continued administration, particularly in the case of opium and hypnotics, the effects of a drug become less evident, and the dose may have to be increased.

Cumulative Action.—The duration of action and the rate of excretion influence the amount of a drug which can be administered with safety in a given time. When the drug acts rapidly and for a short time only the dose may be repeated frequently. On the other hand, if, as in the case of digitalis, the action develops slowly and is more enduring, it is inadvisable to repeat the drug at too short intervals. When drugs are excreted slowly they are apt to accumulate in the body, and their administration may have to be suspended for this reason. A knowledge of pharmacology is the only guide in this matter.

Correction of Flavour, Colour, and Odour

Palatability.—According to Freyberger, who has specially studied the best methods of correcting the tastes of nauseous drugs in view of the necessities of prescribing for children, the most powerful flavouring agents seem to be those derived from oranges, ginger, peppermint, cinnamon, liquorice, and tolu. Perhaps the most generally useful are fluid extract of liquorice, syrup of oranges, and compound tincture of cardamoms. Children, as a rule, dislike peppermint, which, like ginger, is generally palatable to old people. At times, where the administration of alcohol is desired, or at least not disadvantageous, a liqueur of pronounced flavour, such as chartreuse or curaçoa, may render a mixture pleasant. Drugs which are extremely nauseous are best administered in capsules or cachets. Thus it is almost impossible to conceal the taste of paraldehyde or viburnum, though the former is to some extent masked by almond mixture.

The following may be useful for reference:—

Ammonium carbonate, atropine, belladonna, calumba, cinchona, bitters in general, chloral, codeine, digitalis, strophanthus, the scale preparations of iron, guaiacum, hamamelis, morphine, quinine, strychnine, antipyrin, salicylates, salicin, bromides, bicarbonates, liquor trinitrini, nux vomica, and dilute acids are most effectually concealed by *Syrup of Oranges.*

Aloes, ammonium chloride, benzoin, cascara, and strychnine are masked by *Fluid Extract of Liquorice.*

Atropine, ergot, Epsom salts, laudanum, male fern, rhubarb, senna, strychnine, sulphuric acid, tannic acid, and terebene may be rendered more palatable by *Syrup of Ginger.*

Antipyrin, bicarbonates, boric acid, diuretin, iodides, and the liquor hydrargyri perchloridi may be concealed by *Peppermint Water.*

Bicarbonates, camphor, catechu, codeine, kino, and male fern may be disguised by *Cinnamon Water.*

The following are also fairly palatable combinations:—Ammonium bromide with syrup of oranges and chloroform water. Ammonium carbonate or benzoic acid with syrup of tolu. Ichthyol with oleum citronellæ. Butyl chloral hydrate with glycerine and compound tincture of cardamoms. Chloral with syrup of virginian prunes and cinnamon water. Copaiva with mistura amygdalæ. Astringent iron preparations with glycerine. Sulphate of iron with

dilute sulphuric acid and syrup of ginger. Tincture of belladonna or hyoscyamus, or chlorate of potash, with syrupus mori. Menthol and sugar. Cod-liver oil with essence of almonds and lemon. Ammoniated tincture of opium with aniseed water. Phosphoric acid or acetate of potash with syrupus rubi idæi. Castor oil or salicylic acid or dilute sulphuric acid with syrup of ginger and cinnamon water. Senega with syrup of oranges and chloroform water. Spiritus ætheris compositus or spiritus ætheris nitrosi with aqua carui or aqua camphoræ.

Colouring Agents.— It is seldom necessary to add anything to give colour to a mixture, but sometimes, as in the case of apomorphine, which may yield a green solution unless a few drops of hydrochloric acid be added, the colour may with advantage be modified by the addition of liquorice or acid infusion of roses. Unexpected colour reactions are not infrequent in mixtures containing such coal-tar derivatives as antipyrin and antifebrin, hence it is generally advisable to give these in as simple a form as possible.

Odour. — It is comparatively rare for the odour of a drug to be so objectionable as to require concealment. The singularly clinging smell of iodoform may be disguised by Tonga bean or its active principle coumarin ; one grain of the latter may be added to 50 grains of iodoform. Fluid extract of viburnum ought never to be prescribed as such, on account of its offensive smell, which suggests defective sanitation. It is most frequently given in capsule, or in the form of the proprietary remedy known as liquor sedans.

Incompatibility.— Incompatibility may be pharmaceutical, pharmacological, or chemical: of these, the last is by far the most important. In prescribing new and unfamiliar drugs it is extremely easy to write an incompatible prescription, and in many cases no blame can be attached to the prescriber for doing so. As a general rule, such errors are detected by the dispenser in making up the prescription, wherein incompatible combination of drugs to do harm ; more frequently the mixture is inert from the formation of insoluble bodies, or else is in such a form as to be difficult to administer. Practically, the chief dangerous incompatibilities are those in which alkaloids are precipitated, and therefore cease to be evenly distributed throughout the mixture, and those in which cyanides may be formed. Among minor chemical incompatibilities, changes of colour may be mentioned, as, for example, when borax is combined with acid infusion of roses, giving a bright green colour.

Incompatibility is best avoided by simplicity in prescribing, particularly in the choice of the solvent or excipient ; both alcohol and water lose their solvent power for the particular substances each dissolves, in proportion to the amount of the other present. Obviously, a drug should never be prescribed with its test or antidote.

Pharmaceutical Incompatibility.—Water is a solvent for albuminous, mucilaginous, gelatinous, and saccharine bodies generally, and for most inorganic salts. Its power of dissolving these is diminished by the presence of alcohol. Alcohol is a solvent for oils, resins, gum-resins, and balsams, and its power of dissolving these is diminished by the presence of water. Essential oils cannot be dispensed with water in the proportion of more than one minim to the fluid ounce. Fixed oils, oleo-gum-resins such as copaiva, and alcoholic extracts of resinous bodies can be combined with water only by the addition of an excipient to suspend them. Alcoholic tinctures of diverse strengths should not be mixed, and strong tinctures do not combine well with water. The aquæ of the pharmacopœia are already saturated. Compound infusion of gentian is incompatible with infusion of cinchona. Acids should not be prescribed with liquorice, as the glycyrrhizin is precipitated. Water decomposes the glycerine of boracic acid. Salts of alkaloids should not, as a rule, be prescribed with oils or fats ; the alkaloidal base itself should be used.

Table II

	Incompatible with			
Watery infusions and decoctions.	Resinous tinctures and fluid extracts.	Chloroform.	Volatile and fixed oils and balsams.	
Mucilage.	Tannic acid.	Solutions of lead and iron.	Spirit of nitrous ether.	Alcohol, glucosides, oxalates, carbonates.

upon it is customary for him, unless, as is often the case, he can by skilful compounding get over the difficulty, to communicate with the physician in order that the mistake may be rectified. It is also comparatively rare for an

Pharmacological Incompatibility.—It is unnecessary to say much on this head. It is apparent that to prescribe two active drugs which neutralise each other may defeat the aim of the physician, and to avoid this a knowledge

Table III

The drugs in this vertical column are incompatible with	Tannic and gallic acids.	Alkalis.	Alkaloids and their salts.	Acids.	Spiritus ætheris nitrosi.	Metallic salts.	Iodides.						
Tannic and gallic acid . . .		,,	,, (not gallic)	Concentrated mineral	,,	Most metallic salts and oxides		Acetates	Tincture of guaiacum	Albumin	Antipyrin	Mucilage	Glucosides
Alkalis	,,		,,	Acids		,,		Weak salts	Aqua laura-cerasi		Chloral (= evolution of $CHCl_3$)		
Carbonates and carbonic acid .	,,		,, (except codeine)	,, (except HCN)		Bismuth sub-nitrate and metallic salts			Syrup of squills				
Lime water	,,	Alkaline carbonates	,,	,,		Mercurial and other metallic salts		Arsenites					
Alkaloids	,,	Alkalis		Boric and phosphoric acids			,,	Bromides, borax				Oxidising agents	
Quinine salts	,,	,,					,,	Acetates					
Morphine salts . . .	,,	,,			,,	Lead sub-acetate	,,	Borax					Decomposed by codeine
Strychnine salts . . .	,,	,,					Potassium iodide	Bromides					
Free acids and acid salts . .	,,	,,				Metallic oxides, hydrates, and carbonates		Relatively weak or volatile salts	Syrup. ferri phosphatis	Albumin		Tinctures, except in small amount	Glucosides
Dilute hydrocyanic acid . .						Many metallic salts					Antipyrin		
Phosphoric acid . . .		,,	,,			Many metallic salts			Syrup. ferri pyro-phosphatis			Lime water	
Spiritus ætheris nitrosi . . .	,,					Sulphate of iron	,,	Salicylates	Tincture of guaiacum		Antipyrin	Mucilage	Morphine salts

Mercuric chloride	,,	,,	,,	Hydrocyanic acid (= bi-cyanide of mercury)	,,	Lead and iron salts	Arsenites		Albumin	Antipyrin	Soap		
Calomel		,,								Antipyrin			
Silver nitrate	,,	,,		Except acetic and nitric acid			Arsenites and chlorides				Undistilled water		
Arsenites (Fowler's solution)	,,		,,	Acids		Many metallic salts	Hypophosphites in acid mixtures	Mercuric chloride			Lime water		
Iodine and iodides			,,	,,	,,	,,	Chlorates (= poisonous iodates)			Paraldehyde	Starch		
Alum	,,	Alkalis and alkaline carbonates				Lead salts	Tartrates, and alkaline phosphates				Lime water		
Salicylates			,,		,,	Many metallic salts	,,			Antipyrin (deliquesces)			
Digitalis	,,					Iron salts and lead acetate		Cinchona					
Chloral		,,		Dilute hydrocyanic acid			,,			Alcohol (= alcoholate)			
Antipyrin		Sodium hydrate and bicarbonate		Mineral and boric, phosphoric, and hydrocyanic acids		Lead and iron salts, calomel, arsenic	Benzoates			Chloral			
Alkali phosphates			,,	Mineral and acetic acids		Metallic salts							
Guaiacum				,,	,,	,,					Oxidising agents		
Creosote				,,	,,	,,					Oxidising agents		
Codeine	,,	Alkalis, except carbonates	Morphine salts	Mineral acids		Iron, lead, etc.							

of pharmacology is the only guide. In many cases, however, drugs which are in some respects physiologically antagonistic are prescribed together with a definite object, as for example bismuth and rhubarb, when the latter is intended to prevent the constipating effect of the former, and digitalis and iodide of potash, in which the salt is believed to antagonise the action of the digitalis on the vessels.

Chemical Incompatibility.—To avoid this a knowledge of elementary chemistry is required. The more common instances of chemical incompatibility are given in Table III. In addition, the following points should be noted.

(1) Any combination must be avoided which will yield the following insoluble salts:—Hydrates, phosphates, borates, carbonates, tannates, and arsenates of the earthy or heavy metals, or alkaloids; sulphides of the metals; sulphates of calcium and lead; mercurous salts; haloid salts of bismuth, silver, or lead.

(2) Powerful oxidising agents ought not to be combined with substances which are readily reduced; admixtures of such bodies in the dry state may cause an explosion. The following are the chief oxidising and oxidisable substances employed in pharmacy:—*Oxidisers*—Chlorine; strong hydrochloric, nitric, and nitro-muriatic acids; chlorates, hypochlorites, permanganates, nitrates, bromine, iodine, bromates, iodates, oxide of silver, peroxides. *Oxidisable*—Phosphates, hypophosphites, sulphur, sulphides, glycerine, sugar, alcohol, oil, ether, creosote, charcoal, tannin, arsenious acid, cyanides, ferrous and mercurous salts.

(3) Explosions from the evolution of carbonic acid are liable to occur when carbonates are dispensed with acid salts or acids in tightly corked bottles. This is most likely to take place by inadvertently dispensing one of the salts mentioned under (5), or the syrup of squill, with an alkaline carbonate.

(4) Weak acids or bases are decomposed by stronger ones, so that salts in solution exchange their radicles, especially if by doing so an insoluble compound can be formed.

(5) The following salts are acid in reaction:—Alum, bismuth subnitrate, ferric chloride, citrate of quinine and iron, ferrous sulphate, lead acetate and nitrate, quinine bisulphate, zinc sulphate.

(6) Alkaloids are precipitated by alkalis. Iodides, particularly double iodides (*e.g.* potassio-mercuric iodide), bromides, tannic acid, benzoates, and salicylates are likely to produce insoluble salts of most alkaloids. The precipitate is soluble in alcohol. Caffeine is not precipitated by iodides. Acetate of quinine is almost insoluble.

Methods of Administering Drugs

I. *By the Mouth*

(1) *Mixtures.*—In mixtures the drugs are dissolved or suspended in a suitable menstruum, usually water or alcohol. A general acquaintance with the solubility of the various drugs is presupposed; for additional information a table of solubility must be consulted. It is usual to prescribe mixtures containing 12 or 16 doses of half an ounce or two drachms respectively; though convention favours the six-ounce mixture, it will, as a rule, be found more convenient to prescribe four ounces of the smaller dose.

When the mixture contains insoluble substances some excipient should be added to suspend them. The best of these are glycerine, syrup, and gum-tragacanth, which is preferable to acacia for suspending bismuth. One ounce of glycerine may be added to a six-ounce mixture; of tragacanth powder, 1½ to 2 grains to the ounce is enough. It is not generally known that the addition of a few grains of powdered chalk increases the amount of bismuth which can be suspended in a given bulk of fluid. It is thus possible to give 7½ grains of bismuth in a drachm—an advantage in prescribing for children.

(2) *Emulsions.*—These are mechanical suspensions of oils or resins in a state of fine division, giving fluids of the appearance and consistency of cream. The excipients most commonly employed are mucilage, yolk of egg, almond mixture, and alkalis. The last, however, as they act by saponifying part of the fat, should as far as possible be avoided. Tincture of quillaiæ is a suitable excipient for paraldehyde and many oils; for cod-liver oil, yolk of egg is the best, but as the making of a good emulsion is a matter requiring considerable skill, it may to a large extent be left to the pharmacist. Moreover, many of the drugs which are given in this form may equally well be dispensed in capsule. Acids, metallic salts, and large quantities of alcohol are inimical to the process of emulsification.

(3) *Pills.*—Drugs which are nauseous, or insoluble, such as extracts or resins, may be ordered as pills, but this mode of exhibition is unsuitable for bulky substances and efflorescent or deliquescent salts. Thus administered, drugs act slowly, often only after passing out of the stomach. In prescribing pills it is perfectly legitimate to leave to the compounder the choice of the excipient—far better to do so than to order an unsuitable one. Nevertheless, the following hints may be of service to those who prefer to give the directions themselves.

Confection of roses is adapted for dry, heavy powders, and generally when a somewhat pasty excipient is wished. *Glycerine of tragacanth* and *syrup of glucose* are very widely useful; so also are *vegetable extracts*. *Liquorice powder*, or *liquorice* and *compound tragacanth powder*, are suitable when moisture is required to be absorbed. *Soap*, or *soap with magnesia*, forms a good pill mass with many essential oils. Resinous extracts, gum-resins, etc., may be

formed into pills with the addition of a little *alcohol*. Creosote may be made into a pill with breadcrumb, and potassium permanganate or nitrate of silver with kaolin.

After being prepared, pills are coated in various ways, but this is purely a pharmaceutical matter. When it is desired that the drug shall act only in the intestine the pill should be coated with salol or keratin.

(4) *Powders.*—Insoluble substances, and those which would be incompatible with each other in fluid form, may be ordered as powders. If only a very small dose of a drug is given—*e.g.* ¼ grain of calomel—the powder should be brought up to a convenient bulk by adding some inert substance such as milk sugar. Small powders may be given dry on the tongue or in a spoonful of milk; otherwise it is well to order them to be dispensed in cachets, which have almost entirely superseded wafer papers.

Cachets consist of two watch-glass shaped pieces of wafer paper, the margins of which cohere on being moistened. They are adapted for insoluble or nauseous powders which it is inadvisable to dispense in fluid form, such as quinine, phenacetin, salacetic acid, salol, saccharated carbonate of iron, etc. They should be moistened before being swallowed. Quantities up to 20 or even 30 grains of powder can be dispensed in a cachet.

Tablets.—Many powders can be conveniently prescribed in tablets, and a number of pharmacists are now able to dispense these according to any desired formula. It should be noted that the word "tabloid" is a proprietary trade name, and if it be used the preparations of the firm which invented it must be supplied. When a prescription is to be made up in tablet form the direction to the dispenser is "Dispense in tablets," or "Fiat tabella. Mitte tales . . ." Hygroscopic or deliquescent salts should not be prescribed in tablets; the average amount which can be conveniently compounded thus is from 5 to 10 grains.

Chocolate, known in pharmacy as *pasta theobromatis*, is, apart from its flavour, a very suitable basis for some tablets. The solvent action of its fats renders it useful for fixing nitroglycerine, menthol, and alkaloids, also for pepsin, santonin, bismuth, and iron (Martindale).

(5) *Capsules.*—Gelatine capsules are now largely used as a means of concealing the taste of unpleasant drugs. They are made to contain amounts up to a drachm, but except for castor oil such large sizes are seldom used. In most cases the dose is 5, 10, or 20 minims. Not all drugs, however, can be dispensed in capsules. Thus any solid which is at all hygroscopic will absorb water from and burst the gelatine covering it. Again, watery fluids cannot be retained in a capsule. When, as is often the case, such nauseous preparations as watery solutions containing viburnum are ordered in this form, the pharmacist has to evaporate the mixture before filling the capsule. This necessitates loss, and enhances the cost considerably. On the other hand, all forms of oil, and drugs insoluble in water may appropriately be prescribed in capsule.

(6) *Granular Effervescent Preparations.*—Several of these are official, but there are in addition a number of salts which may be prescribed in this form. Some of these are—antipyrin, Carlsbad salts, the citrates of caffeine, iron, lithium, magnesium, and others; the salicylates of lithium and sodium, salicylic acid, etc. The dose of all is about a drachm.

(7) *Confections.*—The use of confections is somewhat limited, but they should not be forgotten in prescribing for children. Aperients such as manna and sulphur, hæmatinics such as reduced iron, and other drugs in which a certain laxity of dosage is permissible, may be given with confection of roses or honey.

II. *By Mucous Membranes in General*

A. Throat and Respiratory Tract.—(1) *Inhalations.*—These are given by adding the desired quantity—about half a drachm—of any volatile drug to a jug of boiling water, and telling the patient to respire the vapour. Special inhalers may also be used. Among the commoner drugs employed in this manner, creosote, carbolic acid, terebene, pine oil, iodine, and friars' balsam may be enumerated. It is sometimes (*e.g.* in the case of menthol) advisable to use an alcoholic solution of the drug in this way.

(2) *Vapours* constitute another means by which the same end can be attained. A small quantity of a volatile substance, such as crude phenol, is placed in a metal evaporating dish, and gently warmed by a spirit-lamp or night-light. The vapour is thus allowed to escape continuously into the air of the sick-room. A vaporiser of this description is chiefly used in lung diseases with fœtid expectoration, and in whooping cough.

Similar drugs may be employed at ordinary temperatures by dropping them on a suitable respirator worn by the patient. A method of inhalation by compressed air has been employed in pulmonary phthisis. The patient sits in a cabinet into which air laden with the vapour of creosote is pumped under pressure.

Fumigations, or inhalations of medicated smoke, are much used in the treatment of asthma.

(3) *Gargles* are watery solutions of drugs used for the sake of their local action on the throat. The average quantity employed on each occasion is ½ to 1 ounce. They usually contain alkaline, antiseptic, or astringent drugs, or combinations of these. Rose water may be used as a vehicle to render them more palatable, and some glycerine added to increase their viscosity.

(4) *Sprays* are liquid preparations which are used in an atomiser for application to the upper air passages. The vehicles chosen are usually distilled water, glycerine, oil, or liquid paraffin, while menthol, cocaine, and carbolic acid are among the most common of the active substances selected.

(5) *Insufflations* are medicated powders which are either snuffed up the nostrils or introduced into the mouth, nose, or larynx by means of a suitable insufflator.

(6) *Pigments.*—This name is given to thick liquid preparations which are brushed on the throat. Glycerine is the most common vehicle, but oils and water may also be used.

(6) *Trochisci* and *Pastilles.*—A number of lozenges are officinal; should it be desired to prescribe a drug not included in these in this particular form, it is sufficient to indicate the quantities, and write the direction "*Ft. Trochiscus.*" Lozenges are of a hard consistence and dissolve slowly in the mouth. When a softer form is desired, the quantity of fruit paste may be increased, or a pastille may be ordered. The basis of pastilles is a flavoured glycerine-jelly (glyco-gelatin). It will, however, be found that the taste of alkaloids and bitter drugs is scarcely concealed by this basis, though menthol may be made palatable in this form. Glyco-gelatin is incompatible with drugs containing tannic acid.

(7) *Intra-laryngeal and Intra-tracheal Injections.*—This method has been adopted with the object of bringing drugs into direct contact with the respiratory passages and lungs. A special curved syringe is used, and, at least until considerable dexterity is attained, the injection is made with the aid of the laryngoscope. Antiseptic drugs in oily solutions are generally introduced.

B. Rectum.—(1) *Enemata.*—Injections into the bowel are given (1) as a means of introducing drugs into the system in unconscious persons or when for any reason stomach or subcutaneous administration is undesirable; (2) to produce a local action; (3) to supply nourishment.

In the case of the first group the enema should be small, not as a rule exceeding two ounces; the dose of the active agent is about double that required when it is given in the usual manner.

Enemata which are designed to act locally are usually given either to evacuate or cleanse the bowel, or to remove flatus, to act as sedatives or astringents and check diarrhœa, or to destroy parasites. Evacuant enemata are large, and consist of water or soap and water in which olive oil, castor oil, or turpentine may be suspended. Sedative enemata should not exceed one or two ounces; they may consist of thin starch, gelatine, or mucilage containing half a drachm of laudanum. Large injections of astringents are sometimes used in cholera and diarrhœal diseases generally. As it is desired that these should be retained as long as possible, they should be injected very slowly and steadily, the buttocks of the patient being raised so that the fluid may gravitate into the colon. Anthelmintic enemata may measure about half a pint for an adult; the preparations most frequently employed are salt (2 drs. to 10 ozs.), infusion of quassia, tincture of perchloride of iron (1 dr. to 10 ozs.), or thymol.

(2) *Suppositories.*—The best basis for these is cacao butter, with which the drugs to be administered are to be incorporated. Glycerine jelly may also be used. The irritation it produces depends on the proportion of glycerine it contains; gelatin lessens this, or a sufficient quantity of water annuls it completely.

Attempts have been made to utilise the rectal mucosa as a channel for the absorption of various drugs in gaseous form—*e.g.* chloroform vapour, ether vapour, hydrogen sulphide, carbonic acid, etc.—but these have not met with any degree of success.

C. Genital and other Mucous Membranes.—In this case drugs are used for their local action only, though it must be remembered that certain general effects may ensue. Thus we once noted a quite definite development of the drug-habit following the prolonged use of strong cocaine ointment for intractable pruritus.

In these regions the drugs employed are generally introduced in the form of watery injections, pigments, pessaries, or bougies. *Pessaries* are prepared with a basis of glycerine jelly where a hygroscopic effect is desired, or of cacao butter. *Pigments* may either be applied with a brush or swab, or on tampons of wool which are allowed to remain *in situ*. *Urethral bougies* are made with a gelatin or cacao butter basis.

Nasal and *aural* bougies and suppositories, of similar composition, are also occasionally prescribed.

III. *By the Cutaneous Surface*

In the majority of cases drugs are applied to the skin with the object of treating some cutaneous disease by local action. Before considering the different modes by which this can be carried out, one or two methods which do not fall into the above category may be mentioned.

Counter-irritants are generally employed in order to influence some deeply seated organ. Of those in common use, the principal, arranged in order of increasing potency, are mustard, turpentine, liniment of ammonia, tincture and liniment of iodine, croton liniment, and cantharides preparations. The milder liniments should be rubbed on with the hand; iodine and croton liniment should be painted on with a brush. The last is somewhat uncertain in its action; it should in the first instance be diluted with an equal bulk of olive oil or soap liniment, and should never be applied to exposed parts of the

skin, as it may cause scarring. Cantharides may be ordered as the plaster, to be applied at night and kept on for six or eight hours, and followed by a linseed poultice if need be. The liquor epispasticus may also be used. Great care ought always to be taken in the use of strong counter-irritants in children, since in conditions of debility, such as follow acute diseases, particularly measles, sloughing of the skin readily takes place.

More powerful than any of the preceding are *Escharotics* such as lunar caustic, strong nitric acid, caustic potash, etc.

The skin is rarely used as a channel for the administration of drugs intended to act generally. Watery solutions penetrate its horny layer with great difficulty, unless carried in by the galvanic current—a method of medication which, after falling into almost complete desuetude, has recently been revived with considerable success as a means of obtaining a more or less local action—*Electrolysis*, or *Cataphoresis*. Only electrolytic solutions can be employed in this way; drugs which do not ionise cannot be introduced into the body by electrolysis. Salts in solution dissociate into an electro-positive metallic ion (kation) and an electro-negative acid radicle ion (anion) (*vide* art. "PHARMACOLOGY," vol. vii. p. 443). When a current is passed through such a solution the kations descend, the anions ascend, the current. The human body is an electrolyte, and if an electrolytic solution be used at the electrodes of a battery connected with the body the descending kations will penetrate the skin at the positive pole; the ascending anions will enter at the negative pole. This can be shown experimentally in animals: strychnine salts cause poisoning when applied at the positive, but not at the negative electrode, the kation here being the active ion; with potassium cyanide the reverse is the case, the anion being toxic. The rule therefore is, to use the positive pole when the action of the metallic ion is desired, the negative pole for the acid radicle. As examples we may mention the use of zinc chloride at the anode for rodent ulcer; salicylate of soda at the cathode for chronic rheumatism. In the one case the zinc, in the other the salicylic ion, acts locally. The solutions are applied on layers of lint connected with the electrode; the other electrode is placed on some indifferent part of the body. A current of 30 or 50 ma. more or less is used. (Dawson Turner, *Ed. Med. Jour.*, 1908, vol. xxiii., N.S.)

Apart from this, if the absorption of a drug through the skin is desired, it must be applied in the form of an ointment. *Fumigation*, as a means of attaining the same end, has fallen into almost complete disuse, and the possibility of a powder being absorbed by the sebaceous and other glands is not made use of in practice. The same remark applies to the *Endermic method*, in which powdered drugs were applied to and absorbed from the raw surface left by a rapidly acting vesicant, such as strong ammonia or chloroform.

In fact, the chief drug which is now administered by the skin is mercury in the inunction treatment of syphilis. The blue ointment is thoroughly rubbed into the skin, or spread on a binder which is kept applied over a considerable area. More recently the cleaner practice of wearing a bib impregnated with mercury has been introduced. Another drug which is sometimes given by this channel is guaiacol, which is a powerful depressant and antipyretic when applied to the skin in fever.

Local Applications to the Skin.—(1) *Ointments.*—The most common bases are lard, vaseline, lanoline, wax, and cetaceum. *Lanoline* (hydrous wool fat, adeps lanæ hydrosus) is readily miscible with water, and in its anhydrous form is distinctly hygroscopic and may therefore irritate. It is somewhat tough, and may be made more workable by the addition of olive oil or vaseline. *Vaseline* is believed to give up incorporated drugs more readily than the others, hence yields a more active ointment. *Oleic acid* is chiefly used for preparing alkaloidal ointments.

Cold creams, or ointments containing a considerable quantity of water, have a refrigerant effect—an instance is the unguentum aquæ rosæ of the pharmacopœia.

When a large proportion of powder is incorporated with a fatty basis a *Paste* results—as in the case of the well-known Lassar's paste, which consists of equal parts of zinc oxide, powdered starch, lanoline, and vaseline. Pastes differ from ointments in being less penetrating, and in permitting excretions to escape more freely.

Salves are ointments medicated in various ways, and spread on muslin. Sometimes the muslin is made impermeable (and the salve more active) by being coated with gutta-percha.

(2) *Plasters*, with one or two exceptions, such as the emplastrum belladonnæ, are not much used. They consist of an adhesive basis, to which drugs are added, spread on leather or linen. Among the best known are the salicylic acid and creosote plaster used in the treatment of lupus; emplastrum hydrargyri, sometimes used for dressing syphilitic ulcers; and the emplastrum plumbi, with which chronic inflammatory conditions are occasionally strapped. The last is a useful plaster basis, to which other drugs may be added.

(3) *Varnishes.*—These are preparations which, when applied to the skin, evaporate and leave a thin adherent coating. They may be medicated in various ways. Among the most generally employed are the linimentum exsiccans (tragacanth 5, glycerine 5, water 100); glycogelatine, or glycerine jelly, which requires to be melted before being applied; collodion; and traumaticin (gutta-percha 1 dr., chloroform 1 oz.). Alkalis should not be added to prepara-

tions containing mucilage. Fats can be incorporated with solutions of glycerine. Collodion is chiefly used as a vehicle for salicylic acid, and traumaticin for chrysarobin. Ichthyol, sulphur, zinc oxide, etc., may be added to the gelatine preparations.

(4) *Dusting Powders* may be applied by dredging, or by laying a muslin bag containing them over the affected part. They act as sedatives to inflamed cutaneous surfaces, and absorb secretion. The substances most commonly used are starch, zinc oxide, boric acid, talc, fuller's earth, kaolin, and carbonate of magnesia. Boric acid alone is irritating, and should be diluted with starch.

(5) *Lotions.*—The basis of lotions is water, to which rectified spirit, glycerine, mucilage, and occasionally oil, may be added. The first of these, by its evaporation, imparts a cooling character to the lotion; glycerine and mucilage are added in order to suspend powders. It should be remembered that some skins do not stand glycerine well on account of its hygroscopic properties.

(6) *Liniments* are generally used as counter-irritants; occasionally as sedatives. They are usually solutions or emulsions of drugs in oils, soaps, or alcohol. Oils and fats penetrate the the skin with difficulty; this is diminished by the addition of alcohol or acetic acid. Soap and yolk of egg are commonly employed as emulsifying agents.

(7) *Baths.*—These may be simply sedative, when such substances as starch (1 to 2 lbs.), bran (2 to 5 lbs.), or gelatine are added to 25 or 30 gallons of warm water. Again, they may be medicated by the addition of alkalis, such as carbonate of soda (2 to 10 oz.), potassium sulphide (2 to 4 oz.), or tar. Mustard baths (1 oz. to 1 gallon) are very powerful stimulants; the patient is held in the bath until the arms of the attendant begin to tingle.

(8) *Soaps.*—These are not much used for the application of drugs to the skin. The intensity of their action is increased if the lather is allowed to dry on, and still more if it is thoroughly rubbed in until dry. One of the most valuable soap preparations is *Hebra's soap spirit*, consisting of two parts of potash soap to one part of rectified spirit, which is of great use in the treatment of seborrhœa of the scalp.

In ordering soaps the chief points to be borne in mind are, that when a decided detergent effect is desired, an alkaline soap should be selected; when all irritation must be avoided, an over-fatty one. It is, however, to be remembered that much of the irritation which some soaps cause is due less to their alkalinity than to the rosin and fats used in their manufacture.

IV. *Injection through the Skin*

(1) *Hypodermic Injection.*—The conveniences of this mode of administering active drugs are many and obvious: the medicine acts rapidly and certainly, it does not derange the digestion, and it can be given to an unconscious or rebellious patient. The introduction of the tablet form of drug for hypodermic medication renders an error in dosage practically impossible, and minimises risk of sepsis; it also renders unnecessary the addition of any preservative to a solution for hypodermic injection. Vaccines consisting of dead bacteria, solutions of tuberculin, etc., should be preserved in sterilised bottles closed by sterile rubber caps, through which the hypodermic needle is thrust to withdraw the requisite dose. Some hypodermic preparations—*e.g.* ergot, adrenaline—may be conveniently preserved in hermetically sealed glass capsules, each containing a single dose. Provided the needle is kept clean there is very little risk of suppuration, but it is safer to use a syringe and needle which have been boiled before use. In giving the injection, a fold of skin is pinched up and the needle introduced through the whole thickness of the true skin, so that the fluid can be painlessly injected into the loose subjacent cellular tissue. If the needle does not penetrate sufficiently, and the fluid is injected into the deeper layer of the epidermis, some pain will be caused. After the needle is withdrawn the part ought to be gently rubbed to promote absorption of the fluid. It should be remembered that a hypodermic injection of ether is much more painful than one of an ordinary alkaloidal solution, and that, particularly when the vitality of the patient is low, a superficial slough may occur.

In giving a subcutaneous injection, the neighbourhood of large veins and nerve trunks should be avoided. When, however, local anæsthesia over a considerable area is aimed at, drugs such as cocaine or morphia are sometimes injected near to, or actually into the sheath of, a nerve trunk.

(2) *Intramuscular Injections.*—These are employed chiefly in the administration of drugs such as ergot, which are reputed to be liable to cause an abscess, or which, like calomel or mercury in oily suspension, are intended to be very slowly absorbed. Muscles of coarse fibre, such as the glutei, should be selected as the point of injection.

(3) *Intravenous Injection.*—This mode of adminstration, on account of the obvious risks attending it, has never enjoyed a great vogue. Drugs introduced into veins act much more rapidly than when injected under the skin. The principal applications of intravenous injection are for the introduction of saline solution to cope with sudden collapse, and of alkalis in diabetic coma and acidosis generally. Among special uses of this method, the injection of mercury in syphilis, of formalin in septicæmia, and of iodoform and other antiseptics in phthisis may be mentioned.

(4) *Injection into the Subcutaneous Tissue* (*Hypodermatoclysis*).—Here also, saline solution is the agent chiefly employed. The fluid may be introduced at almost any part of the body, but sites where the skin is loose and cellular tissue abundant—*e.g.* the mammary region—are to be preferred. A fairly large needle, or a number of needles, should be employed, and the fluid, amounting to one or two pints, allowed to flow in by gravity. The vessel containing it may be attached to the patient's bed about eighteen inches or two feet above the point of injection. Care should be taken to maintain the fluid at a temperature of about 100°. In selecting a site for the injection, one which will interfere with any surgical procedure which may afterwards be necessary should of course be avoided.

Injections of saline solution into the retro-rectal connective tissue have been advised for incontinence of urine. The needle, guided by a finger in the rectum, enters near the tip of the coccyx and is carefully passed up behind the bowel. About a hundred cubic centimetres of fluid may be injected in this way.

The injection of paraffin into the subcutaneous tissue for cosmetic and other purposes does not fall within the scope of this article.

(5) *Intra-spinal Injections.*—Of late years these have been somewhat extensively used, particularly for the induction of cocaine or stovaine anæsthesia below the level of introduction. The strictest antiseptic precautions are naturally essential. A syringe with a needle four or five inches long, which can be boiled, should be used. The patient is placed with the thighs flexed on the abdomen, and the point of the needle is introduced between the third and fourth lumbar vertebræ (just above a line joining the highest points of the iliac crests) about half an inch to one side of the middle line. The needle is thrust steadily in, in a slightly oblique direction upwards and inwards, its point being withdrawn if it catches against the arch of a vertebra. When the needle reaches the spinal canal a few drops of cerebro-spinal fluid flow out; the escape of blood shows that a vein has been punctured. When the needle is in the canal the syringe is attached, and the fluid slowly injected. Some advise that before the solution is introduced an equivalent quantity of cerebro-spinal fluid should be allowed to drain away. When cocaine is the drug selected the patient's head and shoulders should be raised after the injection, so as to prevent the fluid from passing up the spinal canal. In using intra-spinal injections of collargol for cerebro-spinal fever I have found traces of the drug at the base of the brain two days after the drug was injected. Evidently, therefore, substances introduced by lumbar puncture may affect the cranial as well as the spinal contents.

(6) *Epidural Injections* have recently been highly recommended as a means of treating enuresis nocturna. Saline solution is the drug employed. The needle is introduced at an angle of 60° to the skin, just above the middle of the line joining the cornua coccygea of the sacrum. When the resistance of the anterior wall of the sacral canal is felt the needle is inclined parallel with the skin and passed up the sacral canal. From 10 to 40 c.c. of saline solution may be injected. The dura should not be punctured.

Intra-cerebral injections have been used in the treatment of tetanus by the antitoxin, with the object of introducing the serum into the immediate neighbourhood of the poisoned nerve centres.

(7) *Intra-Pulmonary Injections.*—The direct introduction of antiseptics into the diseased lung by means of an ordinary exploring needle has been tried, but is neither free from danger nor attended by decided benefit.

Extra-Pharmacopœial and other New Drugs

In addition to the drugs which find a place in the Pharmacopœia, new remedies are constantly being introduced. For information concerning these, two standard authorities, Martindale and Westcott's *Extra-Pharmacopœia* and the *British Pharmaceutical Codex*, should be referred to. Besides giving a great deal of pharmaceutical information, both volumes contain a number of useful formulæ, many of which are modelled on those of remedies which have gained popularity under various commercial names. As well as giving these formulæ, the compilers of the Codex have introduced brief descriptive names for a variety of substances of definite composition which are already known under special names. Many of these special names are trade protected and can only be legally applied to the products of firms or individuals in whom proprietary rights in these names are vested. Everyone, of course, may produce the drugs under the Codex name, but when the proprietary name is used only the product of the firm which has a legal right to the name can be dispensed.

The advantages of prescribing under the Codex names are: (1) the encouragement of "free trade" in the manufacture of drugs; (2) in many cases, the concealment from a patient of the nature of the remedy; and (3) economy. To give an instance: a certain fashionable drug sells wholesale under its original (trade) name at 1s. 4d. per ounce; the same drug can be supplied, if ordered under its Codex name, at a quarter of the sum. On the other hand, by ordering a drug under its trade name we have such guarantee of its quality as the reputation of its maker affords.

The following list, which is not exhaustive, giving the synonyms of some of the chief com-

mercial products now made, has been compiled from the *Codex*, 1907 :—

Trade Name.	B.P.C. Name.
Acetysal	Aceto-salicylic acid
Actol	Silver lactate
Adnephrine	Adrenine
Adren	,,
Adrenaline	,,
Airoform, Airogen, Airol	Bismuth oxyiodogallate
Albolene	Liquid paraffin
Aletodin	Aceto-salicylic acid
Ammonol	Chiefly acetanilide
Analgen	Quinalgen
Analgesine	Antipyrine
Anodynin	,,
Antikamina	Chiefly acetanilide
Antisepsine	,, ,,
Antitoxiue	,, ,,
Argonin	Silver caseinate
Argyrol	Silver vitellin
Aristol	Thymol di-iodide
Arrhenal	Sodium metarsenate
Aspirin	Aceto-salicylic acid
Atoxyl	Sodium anil-arsenate
Benzosol	Guaiacol benzoate
Betol	Naphthol salicylate
Bromoacetanilide	Chiefly acetanilide
Calcinol	Calcium iodate
Castor oil powders	Magnesium ricinoleate
Chloretone	Chlorbutol
Citramine	Formamol
Collargol	Silver colloid
Creosotal	Creosote carbonate
Cystamine	Formamine
Cystogen	,,
Dermatol	Bismuth subgallate
Dionin	Ethyl morphine hyd.
Diuretin	Theobromin-sodium salicylate
Duotal	Guaiacol carbonate
Eucaine	Betacaine hydrochlor.
Euquinine	Quinine ethyl carbonate
Europhen	Butyl cresyl iodide
Exalgin	Methyl acetanilide
Formin	Formamine
Hæmostasine	Adrenine
Helmitol	Formamol
Hemisine	Adrenine
Heroin	Acetomorphine
Ichthalbin	Albumin ichthyosulphonate
Ichthargan	Silver ichthyosulphonate
Ichthoform	Formaldehyde ichthyosulphonate
Iodol	Iodopyrrhol
Itrol	Silver citrate
Kelene	Ethyl chloride
Largin	Silver albuminate
Laxatin	Phenophthalein
Laxoin	,,
Levuretine	Zymin
Levurine	,,
Neurotropine	Formamol
New urotropin	,,
Nosophen	Iodophenophthalein
Novargan	Silver proteinate
Ol. deelinæ	Liquid paraffin
Paranephrin	Adrenine
Parolein	Liquid paraffin
Phenalgin	Chiefly acetanilide
Pinol	Oil of pine
Proponal	Homalurea
Protargol	Silver proteinate
Pumiline	Oil of pine
Purgen	Phenophthalein
Pyraloxin	Oxidised pyrogallic acid
Renaglandin	Adrenin
Renostypticin	,,
Salazolone	Antipyrin salicylate
Salipyrazolon	Antipyrin salicylate
Salipyrin	Antipyrin salicylate
Saletin	Aceto-salicylic acid
Solurol	Thymic acid
Styracol	Guaracol cinnamate
Suprarenaline	Adrenine
Suprarenin	,,
Tannalbin	Tannin albumin
Tannigen	Acetannin
Tannocol	Tanno gelatin
Tannoform	Methyl ditannin
Tetronal	Ethyl sulphonal
Trional	Methyl ,,
Uretone	Formamine
Urisol	,,
Urophenol	Formamol
Urotropin	Formamine
Vasoconstrictine	Adrenine
Veronal	Malourea
Xaxa	Acetosalicylic acid

Presphenoid. — The anterior part of the sphenoid bone.

Prescription.—The written form in which a physician orders certain medicines to be made up by the chemist for use by a patient. (*See* PRESCRIBING.) In Law, *prescription* is "the undisturbed and continuous enjoyment of a legal right for a period fixed by law, on the expiry of which all attempts to overthrow it are barred"; and, strictly speaking, insanity, by preventing a person knowing that his rights have been invaded, ought to cause suspension of the law of prescription, but it has not always had this effect in practice.

Presenility. *See* PROGERIA.

Presentation.—The relation of the long axis of the fœtus to the long axis of the uterus of the mother, the *presenting part* being the region of the fœtus first touched by the obstetrician's hand during his examination (*per vaginam*) of the woman in labour. *See* LABOUR, DIAGNOSIS AND MECHANISM; LABOUR IN MULTIPLE PREGNANCY (*Frequency of Presentations*); LABOUR, PRECIPITATE AND PROLONGED (*Contracted Pelvis, Mechanism with*); LABOUR, ACCIDENTAL COMPLICATIONS (*Presentation of the Cord*); LABOUR, OBSTETRIC OPERATIONS (*Version*); PREGNANCY, HÆMORRHAGE (*Unavoidable, or Placenta Prævia*).

Preservation of Food-Stuffs.—Various means (refrigeration, salting, tinning, smoking, and the addition of chemicals) are employed to prevent putrefaction in such foodstuffs as meat, fish, milk, butter, vegetables, and fruit; there are laws regulating the use of preservatives. *See* MILK (*Adulteration, Preservatives*).

Pressure. *See* ANEURYSM (*Signs and Symptoms, Pressure Effects*); LABOUR, PRECIPITATE AND PROLONGED (*Contracted Pelvis, Pressure Marks on Fœtal Head*); LABOUR, ACCIDENTAL COMPLICATIONS (*Pressure on Cord in Prolapse*, etc.); LABOUR, POST-PARTUM HÆMORRHAGE (*Pressure on Uterus*); METEOROLOGY (*Atmospheric Pressure*); NERVES, PERIPHERAL (*Injuries, Pressure Lesions*); PULSE (*Arterial Pressure*); ULCERS AND ULCERATION (*Pressure Sore*).

Presternum.—The upper or anterior segment of the sternum; the manubrium.

Preston's Salt. — Carbonate of ammonium. *See* AMMONIUM.

Presystolic.—Preceding the ventricular systole of the heart, or occurring during the auricular systole, *e.g.* a *presystolic murmur*. *See* HEART, AFFECTIONS OF MYOCARDIUM (*Physical Examination, Auscultation*).

Preternatural Labour.—A labour in which some region of the fœtus other than the head is the presenting part; breech cases and cross-births are included. *See* LABOUR, DIAGNOSIS AND MECHANISM.

Prevalidin.—An ointment said to contain camphor, balsam of Peru, oil of eucalyptus, and oil of rosemary, combined with a base ("percutilan"); it is recommended as an inunction in cases of phthisis, bronchitis, rickets, etc.

Preventive Medicine.—The science of prophylaxis or the prevention of disease by

diet, hygiene, vaccination, inoculation, and the like. *See* THERAPEUTICS, SERUM THERAPY.

Prevost's Symptom. — The occurrence, in apoplexy, of conjugate deviation of the eyes and head, which looked toward the side of the cerebral lesion and away from that of the paralysed limbs.

Priapism. — Persistent erection of the penis (Gr. *πριαπίζω*, to be like Priapus) due to stone in the bladder, ascarides, disease of the spinal cord, spleno-medullary leukæmia, etc. *See* BRAIN, CEREBELLUM, AFFECTIONS OF (*Hæmorrhage, Symptoms*); PENIS, SURGICAL AFFECTIONS OF (*Priapism*); SPINE, SURGICAL AFFECTIONS OF (*Fracture, Symptoms*); TABES DORSALIS (*Symptomatology, Genital Organs*).

Prickle-Cell Layer. — The stratum Malpighii of the epidermis. *See* SKIN, ANATOMY AND PHYSIOLOGY (*Epidermis*).

Prickly Heat.—*See* MILIARIA (*Miliaria Papulosa*); SKIN DISEASES OF THE TROPICS (*Caused by Climatic Conditions, Lichen Tropicus*).

Primæ Viæ.—Literally, the first passages, *e.g.* the alimentary canal or the bowels.

Primary.—First in order of time, of importance, or of development; *e.g. primary syphilis* (*see* SYPHILIS, *Acquired, Primary*), *primary amputation, primary lateral sclerosis* (*see* PARALYSIS), etc.

Primiparity.—The state of being pregnant for the first time, the pregnant patient being then termed a *primipara*. *See* PREGNANCY, DIAGNOSIS (*Multiparity and Primiparity*).

Primitive or Primordial.—Early, first-formed, original; *e.g.* the *primitive streak* and *groove* in embryology, *primordial ova* in the fœtal ovary, etc. *See* EMBRYOLOGY; PHYSIOLOGY, TISSUES (*Nerve, Primitive Nerve Sheath*).

Primula Obconica. *See* DERMATITIS TRAUMATICA ET VENENATA (*Causal Agents, Vegetable*); MYIASIS (*Plant Stings*).

Prince's Stain.—A mixture consisting of a basic and two acid dyes in solution (toluidin blue, acid fuchsin, and eosin) used for the differentiation of the nuclei and granules of blood corpuscles, etc.

Prismata Adamantina.—Calcified prisms forming a large part of the enamel of a tooth.

Prisms. *See* NICOL'S PRISM; OCULAR MUSCLES, AFFECTIONS OF (*Abnormal Position, Hyperphoria, Treatment by Prisms*); PHYSIOLOGY, NEURO-MUSCULAR MECHANISM (*Vision*); REFRACTION (*Prisms*); SPECTROSCOPE IN MEDICINE; STRABISMUS (*Treatment*).

Prisoners. *See* MALINGERING (*Motive, Prisoners*).

Private Patients. *See* LUNACY (*Private Patients in England*).

Privies. *See* SEWAGE AND DRAINAGE (*Middens*).

Pro-.—In compound words *pro-* (Lat. *pro*, Gr. *προ*, before) means before in time or in space or in estimation; *e.g.* the *proamnion* (a layer of ectoderm and entoderm of the early embryo), the *proatlas* (a vertebral element occasionally found between the atlas and the occipital bone), etc.

Probability. *See* VITAL STATISTICS (*Probable Duration of Life*, etc.).

Probang.—A flexible rod for sounding the œsophagus or larynx; it may have a sponge on its extremity (*sponge-probang*).

Probe.—A flexible rod, slenderer than a probang, for sounding and exploring internal organs (*e.g.* the uterus, ureters), or wounds (*e.g.* bullet-tracks) or canals. Nélaton's probe has a porcelain tip.

Process.—An outgrowth, prominence, or projecting part; *e.g.* the coracoid process of the scapula, the odontoid process of the axis vertebra, the unciform process of the ethmoid, etc.

Prochownick Diet. — A diet given in pregnancy with the object of diminishing the bulk of the fœtus, of delaying its ossification, and of lessening the tissues which lie in the pelvis of the mother; it is said to be useful in cases of pelvic contraction. The following diet is to be given during the last two or three months of pregnancy:—For breakfast, a small cup of coffee (100 c.c.), about 25 grams of biscuit or bread with some butter; for dinner, any kind of meat, an egg, fish with a little sauce, green vegetables prepared in fat, salad, cheese; supper, the same as for dinner, with 40 to 50 grams of bread and butter; water, soups, potatoes, puddings, sugar, and beer are quite forbidden, and the total daily quantity of fluid is not to exceed 500 c.c.

Procidentia. — Downward displacement or prolapse of some organ or part, such as the rectum or the uterus or the iris. *See* GASTRO-INTESTINAL DISORDERS OF INFANCY (*Prolapsus Recti*); PELVIS, PERINEUM AND PELVIC FLOOR (*Descent of the Floor*); PREGNANCY, AFFECTIONS OF GENERATIVE ORGANS (*Prolapsus or Procidentia Uteri*); RECTUM, DISEASES OF THE (*Procidentia*); UTERUS, DISPLACEMENTS OF THE (*Descent*).

Proct- or Procto-.—In compound words *proct-* (Gr. πρωκτός, the anus) means relating to the anus; *e.g. proctatresia* (imperforation of the anus), *proctocele* (prolapse of the rectum), *proctoccypexy* (fixing the rectum to the coccyx), *proctorrhœa* (discharge of mucus from the anus), and *proctospasmus* (rectal tenesmus).

Proctalgia.—Pain, usually neuralgic in character, in the anus or rectum; proctodynia.

Proctectomy.—Excision of the anus or rectum.

Proctitis. *See* Rectum, Diseases of (*Proctitis*).

Proctodæum.—The posterior part of the cloacal fossa of the embryo. *See* Embryology.

Proctodynia.—Pain about the anus or rectum; proctalgia.

Proctoplasty. — Plastic or reparative surgery of the anus and rectum.

Proctoptosis.—Prolapse of the rectum. *See* Rectum, Diseases of (*Procidentia*).

Proctorrhaphy.—Fixation of the rectum or anus with sutures.

Proctoscope.—A rectal speculum.

Proctosigmoidectomy. — Excision of the rectum and sigmoid flexure.

Proctotomy.— Incision into the rectum, with or without division of the sphincters (*external* and *internal proctotomy*).

Procurator-Fiscal. *See* Medicine, Forensic (*Certification, Scotland*).

Prodromata.—The early or precursory phenomena or signs of a disease or other morbid state (Gr. προδρομή, a running forward or sally).

Product.—In Obstetrics the term product is sometimes given to the fœtus, membranes, placenta, and liquor amnii.

Proembryon.—The first indications of an embryo which exist at the end of the first week of antenatal life *in utero*; the extra-embryonic parts, such as the amnion and the umbilical vesicle, are quite recognisable, but the future embryo is little more than a mass of epiblast cells with, as yet, no primitive streak. (*See* Ballantyne's *Antenatal Pathology*, vol. ii. p. 24.)

Proencephalus.—A teratological type in which there is anterior meroacrania, *i.e.* the frontal bones are defective and the cranial contents protrude in the anterior part of the vault.

Professional Spasms. *See* Neuroses, Occupation.

Profeta's Law.—The law that healthy children born of syphilitic parents are not susceptible to infection with syphilis, and that they can therefore be safely nursed by the mother or by a syphilitic wet-nurse. *See* Syphilis (*In Children, Hereditary*).

Profundus or Profunda.—Deep-seated; *e.g.* the *arteria profunda femoris*, and the *superior profunda* branch of the brachial artery.

Progeria.—Premature old age in association with infantilism (Gr. προ, and γέρων, an old man); geromorphism.

Proglottis. *See* Parasites (*Cestodes, Tœnia Solium*).

Prognathism.—Marked projection of the jaws, with small size of the forehead. *See* Anthropology.

Prognosis.—The forecast of the probable course of a disease, and the estimation of its fatal or favourable termination.

Progressive Muscular Atrophy. *See* Paralysis (*With Atrophy of Muscles*).

Progressive Pernicious Anæmia. *See* Anæmia, Pernicious.

Projection. *See* Ocular Muscles, Affections of (*Paralysis, Faulty Projection*).

Projection Areas. *See* Physiology, Neuro-Muscular Mechanism (*Cerebrum, Flechsig's Projection Areas*).

Prolabium.—The central prominence of the lip, *e.g.* in double hare-lip. *See* Palate (*Hare-Lip, Varieties*).

Prolapse and Prolapsus.—Downward displacement of an organ or structure, *e.g.* the uterus, the umbilical cord or funis, or the iris. *See* Brain, Surgery of (*Hernia Cerebri*); Gastro-Intestinal Disorders of Infancy (*Prolapsus Recti*); Labour, Diagnosis and Mechanism (*Podalic Lies, Prolapse of Limbs*); Labour, Accidental Complications (*Prolapse of the Cord, of Arm, of Foot*); Labour, Obstetric Operations (*Forceps, Indications, Prolapsus Funis*); Pelvis, Perineum and Pelvic Floor (*Descent of the Pelvic Floor, Sacro-Pubic Hernia*); Pelvis, Perineum (*Vaginal Enterocele*); Pregnancy, Affections of Generative Organs (*Prolapse of Gravid Uterus*); Rectum, Diseases of the (*Procidentia Recti*).

Proliferous.—Multiplying or reproducing; *e.g.* a *proliferous cyst* (one in which fresh growths are being produced by the lining mem-

brane), *proliferous catarrh* (a stage in the progress of chronic hypertrophic catarrh of the middle ear), etc.

Prolificacy.—The faculty of reproducing abundantly; the capacity of having large families.

Proligerous.—Containing an ovum or germ, *e.g.* the *discus proligerus* of the Graffian follicle of the ovary. *See* Generation, Female Organs of (*Ovary, Histology*).

Pro-Maternity Hospital.—A hospital or ward in a hospital for the reception of pregnant women suffering from some of the maladies of gestation, *e.g.* hyperemesis gravidarum, albuminuria, eclampsia, and hydramnios; more correctly, *pre-maternity* hospital. (*See Brit. Med. Journ.*, I. for 1901, p. 813; I. for 1908, p. 65).

Prominence or Prominentia.—A projection, elevation, or protuberance; *e.g.* the *genital prominence* (in embryology), the *prominentia laryngea* (or pomum Adami), the *prominentia spiralis* (of the membranous labyrinth), etc.

Promontory.—A projection, usually more marked than a prominence; *e.g.* the *sacral promontory*, the *promontory of the cochlea*, etc.

Pronation.—The position of the arm in which the palm of the hand is directed backwards, and the radius crosses over in front of the ulna. The muscles which by their action throw the arm into this position are called *pronators*; it is the converse of the position of *supination.*

Pronephros.—The primitive kidney, a tubular body opening into the Wolffian duct; it is doubtfully represented in the human subject.

Pronucleus.—The nucleus of the ovum after maturation is termed the *female pronucleus*, while the portion of the spermatozoon which enters the ovum in impregnation is named the *male pronucleus.*

Pro-œstrum.—In female animals the stage of preparation for the œstrus is termed the pro-œstrum; it is regarded as corresponding to menstruation in the human female.

Proof Spirit. *See* Alcohol (*Spirits*).

Prootic.—In front of the ear, *e.g.* the *prootic centre* of ossification of the temporal bone.

Propane.—A hydrocarbon (C_3H_8) of the paraffin series; methyl-ethyl.

Propepsin and Propeptone. *See* Physiology, Food and Digestion (*Gastric.*)

Prophase.—The name given to the first group of phenomena in mitosis (division of the centrosome, etc.); it is followed by the *metaphase*, the *anaphase*, and the *telophase.*

Prophylaxis.—Preventive medicine, or the prevention of disease by hygienic and medicinal means. *See* Cholera Epidemic (*Prophylaxis*); Lung, Tuberculosis of (*Treatment, Prophylactic*); Muscles, Diseases of (*Trichiniasis, Prevention*); Scurvy, Infantile (*Treatment*); Smallpox (*Treatment, Prevention*); Typhoid Fever (*Treatment, Prophylaxis*); Yellow Fever (*Prophylaxis*).

Propionic Acid.—One of the acetic series of acids, being ethyl-formic acid ($C_3H_6O_2$), produced by the oxidation of propyl alcohol; *propione*, or diethyl-ketone ($C_5H_{10}O$), is used as a hypnotic and sedative.

Proprietary Foods and Medicines.—Patented or trade-marked foods and drugs. *See* Infant Feeding (*Proprietary Foods*); Prescribing.

Proprioceptive. *See* Physiology, Neuro-Muscular Mechanism (*Action of the Neural Arcs, Receptors, Varieties*).

Proptosis.—Prolapse or protrusion, *e.g.* *proptosis oculi* (exophthalmos). *See* Prolapse.

Propylamine.—A volatile oil (C_3H_9N or $C_3H_7NH_2$) obtained from the destructive distillation of animal matters (bones, the cultures of the bacteria of fæces); it has been used in the treatment of rheumatism.

Pro re nata.—As the emergeney arises, or as circumstances call for; the instructions given when a line of treatment or a medicine is prepared in view of the occurrence of symptoms not yet present. *See* Prescribing.

Proscolex. *See* Parasites (*Cestodes, Tænia*).

Prosector.—One who prepares anatomical dissections for purposes of demonstration to classes of students; he may be a senior student or a graduate.

Prosencephalon.—The fore-brain. *See* Embryology.

Prosopalgia.—Facial pain or neuralgia (Gr. πρόσωπον, the face, ἄλγος, pain). *See* Nerves, Neuralgia (*Trigeminal*).

Prosopo-.—In compound words *prosopo-* (Gr. πρόσωπον, the face) means relating to the face, or facial; *e.g.* *prosopantritis* (inflammation of the frontal sinus), *prosopoterata* (monstrosities of which the face is chiefly affected), *prosopodysmorphia* (facial atrophy), *prosopospasmus* (risus sardonicus).

Prosopus Varus.—Facial obliquity or asymmetry, congenital or acquired.

Prostate Gland.

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The *anatomy* of the prostate gland may be briefly referred to. It is a firm, glandular and muscular organ, weighing a little over half an ounce. About the size and shape of a horse-chestnut, it surrounds the neck of the bladder and the commencement of the urethra in the male. Placed in the pelvic cavity, its base is directed backwards towards the neck of the bladder, while its apex presents forwards. The posterior and larger surface rests upon the rectum, while the anterior and smaller surface is flattened, and is marked by a slight longitudinal groove. The prostate is divided into three lobes—two lateral lobes and one middle—which up to adult life are hardly perceptible. It is perforated by the urethra and the common seminal ducts. The prostate is enclosed in a dense fibrous capsule which is continuous with the recto-vesical fascia and with the posterior layer of the triangular ligament. The substance of the gland is of a pale reddish grey colour, very dense and friable. In structure it is a compound organ, being partly muscular and partly glandular. The muscular elements are of the involuntary kind, and are arranged so as to form an external layer within the fibrous capsule; circular bands also surround the urethra, while fasciculæ extend in all directions through the glandular substance. The adenoid tissue is composed of numerous small saccules, and these opening into elongated canals terminate in excretory ducts, numbering in all from twelve to twenty. These open upon the floor of the urethra, on each side of the colliculus seminalis.

Physiology.—Possessed of two distinct histological elements, the gland has a double function to perform. The glandular structure secretes a milky acid fluid which lubricates the urethra prior to the venereal orgasm, and also forms a portion of the discharge emitted during the sexual act. Its function is like that of the secretion of the vesiculæ seminales, namely, to dilute the seminal fluid, and so, by increasing its bulk, more efficiently convey the semen within the female passages. The muscular elements not only cause the prostatic secretion to be exuded, but also simultaneously assist in expelling the semen from the seminal ducts.

(*a*) *Injuries* of the prostate, except by instruments introduced along the urethra, are not common. The gland may be punctured or lacerated by falls, or by being impaled on spikes entering from the perinæum, or by surgical wounds; but the situation of the gland, deep in the perinæum, as a rule protects it from direct violence. After middle life, on account of the enlargement of the middle lobe, the prostate is liable to injury by the passage of a catheter. This circumstance should always be kept in view, as not infrequently the enlarged lobe is soft and friable, and the employment of force in the use of a metal catheter may readily lead to perforation of this lobe. The *symptoms* of such an injury do not appear immediately, but a few hours after its occurrence the patient may complain of the symptoms of retention of urine, and repeated attempts to empty the bladder may fail to give relief, a few drops of blood only escaping by the urethra. Reintroduction of the catheter may do little good. A small quantity of urine with blood-clot alone coming away. The distension is not reduced, and if prompt measures are not taken symptoms of toxic poisoning rapidly set in, and steadily increase. The tongue becomes dry and rough, the pulse full, rapid, and compressible, respiration is accelerated, the temperature rises, the skin becomes hot and flushed, and the lips livid.

Treatment.—The distension of the bladder must be relieved at once, not by the passage of instruments through the injured urethra, but by supra-pubic incision, with free drainage and irrigation. In wounds from without bleeding may be severe, and demand active treatment. In such cases, provision having been made for the free escape of urine either through a catheter or by supra-pubic incision (but not otherwise), the perineal wound may be plugged. Or the wound may be enlarged and the vessels ligatured. Under no circumstances should sutures be used to close the wound, as such treatment is certain to promote sepsis.

(*b*) 1. *Inflammation of the Prostate* (*Acute and Chronic*).—*Acute prostatitis* arises as a secondary infective process in cases of acute inflammation of the bladder or of the urethra. The most common direct causes are inordinate

sexual excitement, gonorrhœa, and gleet, and when due to gonorrhœa the symptoms of involvement of the prostate most usually appear about the fifteenth day. The other causes are the employment of strong injections, the internal administration of irritating drugs, the passage of instruments, the presence of foreign bodies or of calculi, and mechanical violence applied to the perinæum. The inflammatory process may be checked, or it may go on to suppuration and abscess.

The *symptoms* are frequent and urgent desire to make water, and after micturition the pain, in place of being relieved, is rather increased. It becomes very severe, throbbing, and continuous. The perinæum is hot and tender, there is rectal tenesmus, and pain in the back and loins. Any movement of the bowels causes distress, the anus is swollen, and large hæmorrhoids forming, add materially to the suffering of the patient. Retention of urine, necessitating the employment of the catheter, causes excessive pain. Constitutional disturbance is very marked, and even suppression of urine may be induced in the later stages. The onset of suppuration is indicated by rigors, and its actual occurrence demonstrated by the appearance of pus in the urine; the portion which comes away first containing pus, while in the last few drops blood appears.

Prognosis.—When acute prostatitis is properly treated, and abscess formation is prevented, recovery usually takes place in a short time, but in a few instances the disease may become chronic, and give rise to chronic urethral discharge.

The bowels having been freely moved with four grains of calomel and a saline, the subsequent *treatment* consists of rest, local bleeding, and hot applications to the perinæum. Absolute rest in bed must be enjoined, and from ten to twenty leeches should be applied to the perinæum, which has been previously shaved and thoroughly cleansed. Local bleeding, if carried out *freely*, gives marked relief. Four hours after the bleeding the patient should be ordered a hot hip bath, and this may be repeated every four hours, and during the intervals large soft poultices should be applied to the perinæum, and the patient wrapt warmly in bed. If the pain is very severe a morphia suppository may be given, and if retention of urine occurs, a soft Coudée catheter may be gently passed.

Chronic Prostatitis.—In cases of acute inflammation of the gland the inflammatory products may fail to be absorbed, leaving behind a permanent enlargement of the organ, producing painful and frequent micturition. This form of enlargement is met with in young adults or men in middle life, and is sometimes accompanied by sexual hypochondriasis and diminished power to expel the urine from the bladder. The urine usually contains mucopurulent shreds, and at the end of the flow a few drops of blood may escape. Examination *per rectum* generally shows some enlargement or tenderness of the prostate, and on the introduction of the bougie into the urethra the passage is found to be unduly sensitive, and before the instrument enters the bladder it meets with a distinct sense of resistance in the situation of the neck. The *treatment* consists of the passage of steel bougies, the use of injections, the application of counter irritants, and the administration of tonics. A number twelve Lister's steel bougie should be passed morning and evening. This treatment should be kept up for six weeks if necessary, and at the same time small blisters should be applied to the perinæum. If in this time no distinct improvement has taken place, a solution of nitrate of silver (4 per cent) may be applied to the prostatic urethra, through the endoscope, or other efficient instrument. Three or four such applications may be required. Constitutional treatment must also be employed. In persons of full habit, four grains of blue pill may be given twice a week, and followed by a saline, and a course of tonic treatment should also be recommended. Iron, arsenic, and quinine may be prescribed, and along with good plain diet, pure air, moderate walking, but no riding exercise, and abstinence from all kinds of excitement generally effect a cure.

2. *Periprostatitis.*—In acute inflammation of the gland the infective process may extend to the periprostatic cellular tissue, and when this occurs the disease generally goes on to suppuration, but in a few instances the disease involves the tissue around the prostate in the first instance, and only invades the gland later on.

3. *Abscess of the Prostate.*—Whether or not acute prostatitis culminates in abscess depends upon the virulence of the infective material, the constitutional resisting power of the individual attacked, and the treatment adopted by the surgeon in the early stages of the malady. When left to nature acute prostatitis may result in the formation of an abscess within four days of the onset of the symptoms, or suppuration may be delayed for ten to twelve days. The most prolific cause of abscess is the misuse of the catheter. The formation of an abscess may be suspected when the symptoms of acute prostatitis become suddenly aggravated, accompanied by rigors and a distinct rise in temperature. When the abscess has formed fluctuation may be felt *per rectum* or by pressure on the perinæum. However gently the examination may be conducted it causes the patient great pain. Much suffering is also occasioned during micturition and defæcation. The abscess if left alone may rupture in one of several directions. The pus may infiltrate the perinæum and rupture externally or into the

rectum ; it may escape by the urethra, perforate the wall of the bladder, or penetrating outside the walls of that viscus it may evacuate itself into the cavity of the peritonæum.

Symptoms of acute abscess have been already indicated, but those of *chronic* abscess are often insidious in their onset, and the progress of the disease is slow. Not infrequently the suppuration arises in old neglected cases of stricture of the urethra in cases where the prostate is hypertrophied or where the catheter has been improperly used. Prostatic calculus may also excite the formation of chronic abscess. *Prognosis.*—In acute abscess the outlook is usually favourable, but the formation of a chronic abscess is always a serious matter, and may lead to complete destruction of the gland, which is replaced by a pus-forming sac, and the patient suffers from fever, emaciation, and not infrequently exhaustion from septic poisoning aids to a fatal termination. The *treatment*, as soon as fluctuation can be made out, is free incision in the middle line and drainage. Small incisions opening into the bowel, or aspiration, deserve and meet with no success.

4. *Urinary fistula* not infrequently follows the opening of a chronic prostatic abscess, especially if the patient has an old urethral stricture. These fistulous openings are often difficult to heal. The first indication as to treatment is to relieve the stricture, and when this has been accomplished the patient should be taught to use a soft rubber catheter at regular intervals until urine ceases to escape by the fistula.

(*c*) *Atrophy of the prostate* is occasionally met with in old age, and in those in whom the sexual desires are feebly developed, in eunuchs, and in those who have been deprived of their testicles by disease. The condition does not give rise to any symptoms, and is of little surgical importance.

(*d*) *Hypertrophy of the prostate* pertaining to old age is by a long way the most common and the most important lesion of the prostate met with in surgical practice. Clinically, the enlargement of the gland is the important fact. The special structural features are of secondary concern. The enlargements of the prostate in those of advanced years have been divided pathologically according to the structural changes observed. By far the most serious change in old age is the increase in the muscular elements, together with fibrosis of the organ. This hyperplasia exists in about 30 per cent of men over 60 years old, and is seldom met with in those under the age of 50. Not only may the muscular structures increase in bulk, but in a few instances there may be also an augmentation of the glandular, so that we may have an adenoma of the prostate. The lesion of old age is a fibrosis and muscular hyperplasia in almost all cases.

A true hypertrophy is unknown, but as in all glandular organs so also in the prostate, occasionally adenomata may be found. In so-called "hypertrophy" of the prostate the progress of the disease is slow, and as a rule those who have led sedentary lives are more liable to an attack, but beyond this fact little is known regarding the etiology of the malady. Many predisposing causes have been named,—syphilis, urethral stricture, gout, rheumatism, vesical calculus, etc.,—but their influence as etiological factors has not been proved, beyond the circumstance that they induce a passive hyperæmia of the lower urinary tract. It is a circumstance worthy of note that men who have been deprived of their testicles by operation, accident, or disease, never have enlarged prostates. This enlargement of the prostate differs from hypertrophy of all other organs excepting the uterus in the fact that the onset does not coincide with the period of greatest functional activity of the gland, and an increase in bulk is not due to an augmentation of its active elements. Structurally, the change in many respects resembles a fibrosis due to passive hyperæmia, and therefore is comparable to the analogous enlargement of the uterus (fibro-myoma). The prostate may enlarge uniformly and attain considerable dimensions without causing much discomfort to the patient. On the other hand, without any great general enlargement, there may be a local increase in bulk of the middle lobe only. The effects of this are sometimes very marked. The middle lobe projects upwards, at the neck of the bladder, and may act like a valve to the orifice of the urethra. Or the urethra may be pushed upwards and forwards so that on section the canal is of crescentic form with the concavity looking downwards. When the hypertrophy is general the prostatic portion of the urethra, which normally measures $1\frac{1}{2}$ inches, may be extended in length to 4 inches, and the weight of the gland from being a little over half an ounce may be increased to 10 ounces. When the prostate weighs more than 7 drachms it may be described as abnormal in size.

The *symptoms* of hypertrophy of the prostate depend upon the nature and the position of the new-formed tissue. When the hyperplasia uniformly involves the whole organ a considerable period may elapse between the onset of the disease and the evidence of its presence. While on the other hand if the growth involves the middle lobe only, there may be the occurrence of marked symptoms before much enlargement of the gland can be made out by digital examination *per rectum*. The first indication of increase in the size of the prostate gland is a diminution in the force by which the urine is expelled, then micturition becomes more frequent, and the relief given by the act is incomplete. Should any undue accumulation

occur, as after prolonged sleep, or after taking a large amount of stimulants, the bladder finds difficulty in expelling the accumulated urine. In place of being emptied by one act frequent calls are made within a short period, and even the repeated efforts are not altogether successful. The inconvenience arising from frequent micturition and incomplete relief after the act in voiding the bladder are at first trivial, but as the obstruction increases the functions of the bladder become more and more impaired, in spite of a compensatory hypertrophy of the muscular wall of the bladder developed in order to increase the expulsive force. Sleep is disturbed by frequent calls to pass urine, the efforts to micturate become more ineffective, and the relief to the bladder less complete. That viscus gradually becomes dilated, and habitually fails to empty itself, a portion only of its contents being expelled at each act of micturition; the patient complains of obscure pains in the perinæum, the rectum, and the neck of the bladder. In some instances the pain may extend down one or other great sciatic nerve; he observes that the stream of urine becomes smaller, and the force by which it is expelled gradually less. Micturition does not commence at will; increased efforts occasionally cause escape of the contents of the bowel. As a consequence of continued straining of the parts, hernia, hæmorrhoids, or even prolapse of the rectum may be induced, and passive hyperæmia resulting from increased resistance to the venous return may cause erections unassociated with sexual desire.

If these difficulties consequent upon interference with the mechanism of micturition are not relieved by art, the pressure which has been exerted to void urine induces dilatation of the bladder, and in old-standing cases may cause sacculation of that viscus. Bi-lateral hydronephroses and dilatation of the ureters follow as a natural consequence of the pressure being reflected backwards, but the distension is not always equal on the two sides. On account of these structural changes the functions of the bladder and kidneys show evidence of failure. The bladder remains habitually filled, only the surplus urine escaping at micturition. During the unconsciousness of sleep, to the patient's great discomfort, the urine passes more freely, and he is said to be suffering from "incontinence," a term which may be used if its exact clinical significance is appreciated, but it is essentially faulty in the sense that it does not describe the condition present. The bladder contains too much urine; the "incontinence" applies only to the overflow. The patient's general health steadily deteriorates, and if left unrelieved by surgical treatment acute and critical symptoms may develop at any moment, and may be induced by the most trivial cause. Long-standing urinary embarrassment renders the organs involved specially liable to septic infection, which may take place independently of the use of instruments. But the fact cannot be overlooked that in many instances the symptoms of toxic poisoning closely follow upon the first use of the catheter. After sepsis has been induced the natural course of events is usually very rapid. Let it be accepted that the patient has not been relieved by the surgeon in the early progress of the disease. The prostatic obstruction that induced hypertrophy of the walls of the bladder in the early stage, as the disease advances, also causes dilatation and atrophy of the muscular elements. Following there may also be sacculation of the bladder, dilatation of the ureters and pelves of the kidneys. The urine is acid, pale in colour, deficient in urea, and may contain a little albumin. The patient is feeble, his appetite is poor, and his urinary troubles are as they have been for months. After drinking rather heavily, or after returning from a fatiguing journey, he becomes "chilled," and goes to bed. Early the following morning he finds that he is unable to pass water, and no relief follows the remedies he is accustomed to use. Retention of urine is complete, and for the first time a catheter is passed. In the afternoon and evening he has several severe rigors, his temperature rises rapidly, the pulse is full, bounding, and rapid, the respirations are shallow and augmented in number, the tongue is dry and the lips parched. The skin is moist and soft. The urine now drawn off, in place of being clear and acid, contains some blood, is slightly opaque, and has a pungent odour. Next morning the urine is distinctly alkaline, and contains urea-decomposing organisms, such as staphylococcus pyogenes aureus and S. pyogenes albus. The tongue becomes dry and brown, the skin pale in colour, the quantity of urine secreted is diminished; later on diarrhœa sets in, the patient's temperature rises to 102° or 103° F., and then follow the symptoms of suppression of urine with septic intoxication.

When septic infection occurs at an earlier stage in the disease the progress of events is not so rapid. The symptoms point to subacute cystitis with decomposition of urea prior to evacuation. (*See* "Bladder," vol. i. p. 387.) The urine, from being clear and acid in reaction, slowly becomes turbid. The reaction passes from acid to neutral, then gradually becomes alkaline, offensive in odour, and later on distinctly ammoniacal. Symptoms of cystitis slowly develop, and the urinary difficulties steadily increase, the inflamed bladder being less able to contend with the urethral obstruction. Infection gradually extends from the bladder to the ureters and kidneys. The general health becomes steadily impaired, slight feverish attacks occur at intervals, and toxic poisoning

from septic infection and suppression of urine bring about the end.

The *diagnosis* in the early stage of the disease should depend entirely upon a careful consideration of the symptoms, and a digital examination of the gland through the rectum. This can be done, in the first instance, without the employment of a bougie or sound passed into the urethra. Familiar with the palpation of the normal prostate as regards situation, size, form, and consistence, the surgeon can readily detect by the finger alone a departure from health. On pressing the finger forwards in the middle line, in place of the soft yielding coats of the bladder he finds in hypertrophy of the gland a distinct firmness. Then examining to right and left he can ascertain whether or not the hypertrophy is limited or general, or regular or uniform, whether it is solid or contains fluid. The degree of pain and discomfort which the examination occasions is an important guide. In hypertrophy, unless when there is some rectal complication, the suffering is slight, whereas in enlargement due to inflammation, to malignant disease, or to tuberculous affections, extreme pain will be caused by the pressure of the finger. In acute inflammation the introduction of the finger will be resisted, and the parts will be found soft and heated as well as swollen. The prudent surgeon will avoid examination with the bougie as long as possible, unless symptoms of stone in the bladder demand attention, or the history of the case points to cicatricial stricture of the urethra. In the latter condition the obstruction is lower down than in that produced by an enlarged prostate. It is encountered within 7 inches of the meatus, whereas in hypertrophy the obstruction is beyond 7 or 8 inches, and before a sound can reach the bladder the handle must be deeply depressed between the legs of the patient; moreover, cicatricial stricture is most frequently found in the membranous portion of the urethra, and occurs before or during middle life. In vesical calculus the pain is more severe, is increased at the end of micturition and by active exercise or jolting, diminishes by rest in bed, and is associated with frequent hæmaturia. In calculus the pain and frequent micturition are most troublesome during the day, whereas in hypertrophy of the prostate the urinary difficulties are most marked at night. Phosphatic calculus and enlarged prostate frequently coexist, and the use of the sound can alone clear up the diagnosis. Vesical tumours are attended by intermittent and unprovoked attacks of severe hæmaturia, and in the early stage of the disease not preceded or accompanied by pain unless clots form and obstruct the escape of urine. Fragments of the tumour may be discovered in the urine. In the later developments of the malady the tumour may come to involve the neck of the bladder and impede the passage; pain then becomes a prominent symptom. Examination *per rectum* usually clears up the diagnosis. In malignant disease the swelling is less regular in form, and occasions the patient much pain. Hæmaturia becomes profuse and continuous, anæmia becomes marked, and rapid emaciation occurs, and the inguinal lymphatic glands become enlarged.

In atony of the bladder retention is the marked symptom, but here the catheter passes without meeting with obstruction, and rectal examination shows that the prostate is not enlarged. This is also observed in paralysis of the bladder, but it is always associated with paralysis of the lower extremities.

With respect to *treatment* one important fact must be accepted, namely, that its success or failure depends upon the disease being recognised early and promptly treated. As soon as the enlargement of the prostate prevents the patient from completely emptying his bladder his "catheter life" should begin. The catheter, when used thus early, is a safe and useful instrument, but when its employment is delayed until secondary changes have taken place in the bladder and kidneys, however careful the surgeon may be, its use is attended with much danger. It is often very difficult to make a patient understand that the habitual use of the catheter is necessary in the early stages. The only troubles he has are frequent micturition, a sense of imperfect relief, and some feeling of weight and tenderness at the neck of the bladder. To relieve this, is he to submit himself to the regular use of the catheter? Yes, certainly. Not simply to promote his immediate comfort, but rather to save him from the otherwise inevitable distressing complications which follow unrelieved prostatic obstruction. The treatment must be regulated according as the secondary complications are just beginning or are advanced. Take the former instance first. A patient, a man over fifty years of age, consults the surgeon and complains of frequent micturition and imperfect relief after the act; he has some pain occasionally, but it is never distressing. His general health is good, and beyond the urinary symptoms he has little to complain of. After satisfying himself by a rectal examination that the prostate is enlarged, the surgeon must next ascertain if the bladder is being completely emptied, and this can only be done by passing a catheter. To do this safely certain precautions must be taken in the first instance, that is to say, until the amount of the residual urine is known. To begin with, the surgeon should avoid making an examination in his consulting-room. Send the patient home with instructions to go to bed, and when the surgeon visits him the patient is instructed to pass as much urine as he can by natural effort; then, all precautions having been taken to avoid sepsis, a soft gum-elastic catheter is

gently passed while the patient is in the erect posture. The quantity of water (residual urine) which comes away is a tolerably safe guide in estimating the stage of the disease and the secondary complications to which it has given rise. The patient should be ordered 2 grains of quinine, a hot hip bath, and be instructed to remain in bed till he is seen by the surgeon on the following morning. How often the catheter will require to be used depends upon the amount of the residual urine, but, as a rule, it is well to completely empty the bladder every evening at bedtime. On the other hand, suppose the patient has not sought surgical advice until the disease is far advanced and complete retention has occurred, the method of procedure is somewhat different, and the risks involved are much greater. The greatest care must be taken against septic infection, as all the conditions favourable to rapid invasion are present in the urinary tract. The distended bladder must be emptied very slowly. This can be carried out by a syphon arrangement, by which only 2 or 3 ounces of urine are permitted to escape per hour. The bladder should in this way be completely emptied in twenty-four hours, when the catheter may be withdrawn, unless great difficulty was experienced in introducing it from irregularities of the urethra or from false passages. These circumstances, therefore, alone justify tying in a catheter. The practice, although often convenient for the surgeon, greatly increases the danger of sepsis. In cases where the retention is sudden or attended by acute symptoms, suprapubic drainage must be resorted to. Suprapubic aspiration may be useful occasionally in order to render temporary relief.

The medical treatment consists in the administration of mild laxatives, careful regulation of diet, and the administration of boracic acid or urotropin may be resorted to if there is a tendency to the urine becoming alkaline or if septic infection has occurred. In the latter instance it may also be necessary to wash out the bladder regularly with a mild antiseptic solution, and after the bladder has been cleansed, a drachm of a solution of nitrate of silver (2 per cent) may be introduced and allowed to remain. The patient should be instructed to avoid horse-riding, excess of food or alcohol, sexual excitement, sudden changes in temperature, or undue fatigue.

Radical treatment may be carried out in three different ways—

(1) Castration;
(2) Vasectomy; and
(3) Prostatectomy.

The relationship of the growth of the prostate to the condition of the testicles has been referred to already, and the analogy between uterine fibromyoma and prostatic enlargement has also been pointed out. Keeping these points in view and noting the effect that the operation of oöphorectomy has in reducing the size of the uterine fibroid, Professor White, of Philadelphia, was the first to suggest *castration* with the view to produce atrophy of the enlarged prostate, and thereby restore to natural micturition. Although this method of treatment has been successful in a few instances, it has not proved itself to be uniformly so, and in a certain proportion of cases serious mental derangement has followed complete castration, in some instances amounting to helpless imbecility. In order to avoid the possibility of such a calamity the operation of vasectomy has been proposed and carried out by Guyon and Harrison. The operation is devoid of risk, and the results are said to be satisfactory.

Ligature of the arteries supplying the testicles not only causes atrophy of these glands, but it also induces a decrease in the size of the enlarged prostate. For the operation of vasectomy general anæsthesia is not required, cocain is sufficient. The vas deferens is easily isolated by the finger as it enters the abdominal ring. At this point an incision is made and the vas exposed to view. Each cord should be divided and ligatured, and the whole operation should be performed with as little disturbance to the parts as possible, and with strictly aseptic precautions. The patient is often very restless, liable to displace the dressings and contaminate them with dribbling urine, so it is well to carefully adjust the edges of the wound with sutures, and seal with collodion in addition to dressings applied. The writer has found this mode of treatment very successful, especially in cases of rapid enlargement of the prostate.

Excision of the prostate in whole or in part for the removal of prostatic obstruction and preservation of the expulsive power of the bladder has not proved very successful. Whether prostatectomy is or is not a justifiable operation has been gravely questioned by many surgeons, while only a few advocate its use. Certainly it should not be employed except in cases which have proved intractable to careful and prolonged catheter treatment, and considering the high mortality which attends the operation it should not be undertaken without the patient's knowledge of the risks involved.

The cases which are suitable for prostatectomy are those in which the enlargement is intravesical rather than rectal, and certainly the most favourable of all cases are those where a more or less pedunculated outgrowth has formed in the middle lobe. In such cases prostatectomy is an operation of value, but in sessile growths and in annular formations surrounding the neck of the bladder like a collar little good can be expected from an operation. How is the surgeon to know the exact condition present? A cystoscopic examination is almost impossible; the alternative is suprapubic incision, but this can

be employed only when the bladder is free from cystitis and capable of distension.

Much has been written on the relative advantages of the perinæal, the suprapubic, and the combined methods. The first mentioned has the drawback of giving very little room, and the operation requires to be performed almost entirely in the dark; but, on the other hand, it affords good drainage, it alone is applicable to cases of chronic cystitis with contracted bladder, and it is the most suitable operation when the patient is feeble and a prolonged procedure is inadvisable.

In those cases where the bladder can be well distended the suprapubic operation should be employed. It is a safe one, and should the surgeon fail to reach the growth from the front he may resort to the combined method. Opening the bladder in front, the interior of the viscus may be explored by the finger, and at the same time, using a forehead mirror, reflected light enables the parts to be clearly seen, and admits of the operation being performed with precision and completeness. When the enlargement of the prostate is due to a passive hyperæmia free suprapubic drainage alone gives marked relief; the functional activity of the bladder being placed in abeyance, the nutritive activity of the parts also becomes lessened, and marked diminution of the gland may result.

(*e*) *Simple tumours of the prostate* are very rare. They include the myomata and adenomata. The former resemble in appearance hypertrophy of the gland, and differ in the circumstance that the growth is not limited to those of advanced years. The true adenomata of the prostate is encapsuled and occurs mostly in young men.

(*f*) *Malignant tumours of the prostate*, "carcinoma" and "sarcoma," affect the two extremes of life, childhood and old age. They as a rule appear before the seventh year or after the sixtieth year, but are very rare either as primary or as secondary formations. In childhood the disease is generally sarcomatous in its nature; its growth is usually very rapid, the course of the malady seldom extending over eight or nine months. The carcinomata, on the other hand, are more prevalent in old age. This lesion primarily attacks the glandular structures in one lobe, and from the original centre rapidly spreads until the whole gland is displaced by the morbid growth. It may then rupture the firm fibrous capsule of the prostate and invade the surrounding tissue. This is, however, seldom the case, as usually when left to nature the urinary symptoms are so severe that the patient succumbs before that stage is reached. The symptoms of malignant disease in children are so characteristic that there is little difficulty in distinguishing it from other maladies, but in the disease as it attacks the aged the inexperienced surgeon is apt to be misled. In children the marked feature is persistent and steadily increasing hæmaturia, the blood coming away most copiously at the end of micturition, often at first unassociated with pain, but as the disease advances symptoms of diminished expulsive power combined with those of cystitis declare themselves. In the malignant disease of old age the early symptoms are so similar to those of ordinary hypertrophy that there is much danger of the rarer malady being mistaken for the commoner one. In malignant disease, however, hæmaturia and pain are early symptoms, and the latter is independent of obstruction to the escape of urine. The course of the disease is very rapid, and on rectal examination a hard nodulated mass, which is very tender to the touch, can be detected. Soon the tumour softens and fungates, the urine presents a peculiar, disagreeable odour and becomes septic. Acute cystitis is set up, hæmorrhages are more frequent and more profuse, the symptoms of obstruction rapidly develop, the sufferings of the patient are intensified day by day, anæmia and emaciation become marked, and the patient gradually sinks from exhaustion or dies from toxic poisoning.

Treatment is only palliative; to remove the tumour with success is practically impossible. The administration of opiates in some form, and suprapubic drainage give relief to pain and prolong life.

The suprapubic operation is best performed in two stages. The bladder being first stitched to the abdominal wall, and a few days later, when adhesions have formed, the viscus can be opened and a tube inserted. Catheterism gives rise to great suffering and much bleeding.

(*g*) *Tuberculosis of the prostate* is secondary always to similar diseases of other parts of the urinary tract, the kidneys, the bladder, or the vesiculæ seminales being primarily affected; or it may be due to an infected thrombosis from tuberculous disease of the lungs, of the joints, or of bones. The disease is met with most commonly in early adult life; the morbid process in the prostate being similar to tuberculosis elsewhere. To begin with, the tuberculous infiltration involves one lobe only, and for some time may not produce any very noticeable enlargement, but as the disease extends inflammatory œdema sets in and causes an increase in the swelling. The infiltration is followed by caseous necrosis, which gradually extends towards the mucous membrane of the bladder or bowel. The mass breaks down, the deep, excavated ulcers form, and perforation takes place into the bladder or bowel, or into the ischio-rectal fossa or the peritoneal cavity. These ulcers act as centres of septic infection and rapidly lead to a fatal termination. The *symptoms* during the early stage are not characteristic, especially when the disease attacks the peripheral parts of the gland. Indeed, the onset of the disease is liable to be overlooked,

but its presence should always be suspected when a patient suffering from a strumous enlargement of the epididymis or a tuberculous kidney complains of a weight in the perinæum and rectal tenesmus. The diagnosis may be confirmed by the occurrence of occasional slight hæmorrhages, by the discovery of tubercle bacilli in the urine, and by the presence of an enlarged prostate. The enlargement may consist of one or more isolated masses embedded in the substance of the prostate, or may be due to a general increase in the whole gland. As the disease advances the symptoms become unmistakable. The urine becomes muco-purulent, hæmaturia is a frequent sign, and gradually, as the new formation encroaches upon the neck of the bladder, the symptoms of prostatic obstruction become more marked, and may culminate in complete retention.

The *diagnosis* in the early stages may be difficult in some instances, but during the later developments little difficulty should be experienced. Frequent and painful micturition in a strumous subject associated with occasional hæmaturia, in the absence of vesical calculus, is a strongly suspicious circumstance; and if in these conditions we have muco-purulent urine containing tubercle bacilli, and a painful swelling at the neck of the bladder, in a man between 20 and 40 years of age, the diagnosis of tubercular disease of the prostate may be made with confidence.

The *prognosis* must depend upon a careful consideration of the whole circumstances of the case, the nature and extent of the primary disease, as well as that of the local lesion. Most of the cases run from bad to worse, and acute sepsis of the urinary tract brings about its inevitable termination.

Treatment.—When the local disease is limited and the general constitution good, which it very rarely is in these cases, surgical interference may be justifiable. Some surgeons advocate incising the gland, scraping away the tuberculous material, and packing with iodoform gauze, and no doubt in a few instances this treatment has been remarkably successful. The use of lactic acid and other agents has been advocated, while considerable benefit is also said to be afforded by the injection into the bladder of an emulsion of iodoform in olive oil, three or four times a day. The use of instruments passed into the bladder is to be avoided, as they merely cause irritation and aggravate the disease. In the more severe cases local treatment is practically limited to alleviating suffering by opening, scraping out, and draining the tuberculous abscess, and by administering narcotic drugs in the form of suppositories. Of the constitutional treatment nothing need be said.

(*h*) *Cystic diseases* of the prostate are very uncommon. Of retention cysts there are two varieties: (1) those due to obstruction of the excretory duct of the prostatic follicles, and (2) those caused by closure of the orifice of the sinus pocularis. Besides these there are hydatid cysts which are not strictly prostatic, having their origin in the tissues between the bladder and rectum, rather than in the substance of the gland. By their proximity they may closely simulate prostatic enlargement. These cystic conditions are of pathological interest, but not of much clinical importance, except in so far as concretions are liable to form in them.

(*i*) *Prostatic concretions* are very common in old persons. When small in size and of recent formation they are pale in colour, but if large and old they are reddish brown or black. They vary in size, some being very much like rounded grains of sand, while others may be as large as poppy seeds. They are round or oval in form, and sometimes on section present a stratified appearance and possess a central cavity. These concretions are composed of organic matter derived from the prostatic secretion, and give the reaction of amyloid material. When old they may become calcified and invested in a deposit of phosphate and carbonate of lime, and then form prostatic so-called "calculi." They vary in number from a few to two or three hundred in one prostate, and some by passing into the urethra escape with the urine.

They rarely cause any *symptoms* unless their presence gives rise to inflammation of the gland. In a few instances, however, they have caused irritation at the neck of the bladder, hæmaturia, and obstruction to the flow of urine. When a sound is passed over the prostatic urethra a creaking sensation is felt.

The *treatment* consists in removing the calculi with urethral forceps, or, if this cannot be done, by a median perineal incision, care being taken to injure the gland as little as possible. This operation is resorted to only when the calculi are numerous or large.

Prostatectomy.—Excision of the prostate gland in whole or in part. *See* Prostate Gland (*Hypertrophy, Treatment*).

Prostatitis.—Inflammation of the prostate. *See* Prostate Gland (*Inflammation*); Rectum, Diseases of the (*Rectal Symptoms from Prostatic Disease*); Spermatorrhœa (*Causes*).

Prostato-cystitis.—Inflammation of the prostate and bladder. *See* Prostate Gland (*Inflammation*).

Prostatorrhœa.—The discharge of prostatic fluid, in excessive amount, from the urethra.

Prostatotomy.—Incision into the prostate gland; it may be done with the galvanic cautery (*thermo-electric prostatotomy*).

Prosthesis.—The correction of defects in development or the results of injury by artificial means; literally, "replacing." *See* Mouth, Injuries and Diseases of the Jaw (*Operations on the Jaws, Immediate Prosthesis*).

Prostitutes. *See* Lunacy (*Inveterate Prostitute*).

Protagon.—A glucoside ($C_{160}H_{308}N_5PO_{35}$?) found in nerve tissue, especially in the white sheath, and yielding stearic acid. *See* Physiology, Tissues (*Nerve, Chemistry*).

Protamine.—An organic base ($C_{16}H_{32}N_9O_2$) found in semen, *e.g.* in the heads of spermatozoa, where it is linked with nucleic acid. *See* Physiology, Protoplasm (*Products of Decomposition*).

Protargol.—A protein compound of silver, forming a yellow powder soluble in water, and containing 8 per cent of silver; it is used in ophthalmia neonatorum and in gonorrhœa. *See* Conjunctiva, Diseases of (*Ophthalmia Neonatorum, Treatment*).

Protean.—Taking many forms; *e.g.*, syphilis is a protean disease. The term is derived from the sea-god Proteus, who was said to have the power of assuming different shapes.

Proteates. *See* Physiology, Protoplasm (*Conjugated Proteins*); Physiology, Food and Digestion (*Gastric Digestion, Proteolytic Period*).

Proteids.—The "chief substances" (Gr. πρώτος, first) of animal and vegetable tissues, containing carbon, hydrogen, nitrogen, sulphur, oxygen, and some inorganic salts; examples are found in albumen, globulin, casein, peptone, etc.; their chemical composition is very complex; albuminoids, proteins. *See* Diet (*Nutritive Constituents, Organic*); Digestion and Metabolism (*Absorption of Proteids*); Liver, Physiology of (*Functions, Proteids*); Physiology, Protoplasm (*Chemistry, Proteins*); Physiology, Blood and Lymph (*Chemistry of Blood*); Physiology, Food and Digestion (*Gastric Digestion, Proteolytic Period*); Physiology, General Metabolism (*Protein Metabolism*).

Proteins. *See* Proteids and *Cross References.*

Protene. *See* Diet (*Milk and its Products, Caseinogen*); Invalid Feeding (*Milk, "Protene"*).

Proteolytic.—Causing proteolysis (*i.e.* the conversion of proteids or proteins into diffusible substances such as peptone by the action of ferments called proteolytic ferments). *See* Physiology, Food and Digestion (*Gastric Digestion, Proteolytic Period*).

Proteoses.—Bodies intermediate between proteins and peptones; *e.g.* heteroproteose, deuteroproteose. *See* Physiology, Protoplasm (*Classification of Proteins*); Physiology, Food and Digestion (*Gastric Digestion, Proteolytic Period*).

Proteosoma. *See* Malaria (*History*); Parasites (*Protozoa, Hæmosporidia*).

Proteosuria.—The presence of proteoses in the urine.

Proteus. *See* Micro-Organisms (*Bacteria, Proteus*).

Prothrombin. *See* Physiology, Blood and Lymph (*Coagulation, Enzymes*).

Proto-.—In compound words *proto-* (Gr. πρῶτος, foremost) means first or primitive; *e.g.* a *protoblast* is a cell without a cell wall, and a *prototype* is an original type. In Chemistry *proto-* is a prefix indicating the lowest of a series of compounds; *e.g.* a *protochloride* is that one which contains the smallest relative amount of chloride.

Protopathic.—Idiopathic, as opposed to deuteropathic. *See* Physiology, Neuro-Muscular Mechanism (*Thermal Sense, Protopathic Sensibility*); Sensation (*Head's Experiments*).

Protoplasm.—*See* Physiology, Protoplasm.

Protoproteoses. *See* Proteoses and *Cross References.*

Prototypes. *See* Tumours (*Oncology, Law of Prototypes*).

Protovertebræ.—The primitive segments of the embryo. *See* Embryology.

Protozoa. *See* Parasites (*Protozoa*); Herpes (*Herpes Zoster, Pathology*); Micro-Organisms (*Protozoa*).

Protracted Labour. *See* Labour, Precipitate and Prolonged.

Protuberance or Protuberantia.—A projection or prominence, *e.g.* the *occipital protuberance* and the *protuberantia laryngea.*

"Proud Flesh."—Exuberant granulation tissue such as is seen in the weak condition of an ulcer. *See* Ulcers and Ulceration (*Classification*).

Proust-Lichtheim Test. *See* Aphasia (*Clinical Features, Cortical Aphasia*).

Proximate Principles.—The substances in food which are of the same nature as the materials found on the analysis of dead protoplasm. *See* Physiology, Food and Diges-

TION (*Nature of Food, Food-Stuffs yielding Energy*).

Prozygosis.—The teratological type in which two fœtuses are joined more or less closely by the heads into a double monster, the trunks being more or less separate; syncephaly.

Prune-juice Sputum. *See* EXPECTORATION (*Pneumonia*); PNEUMONIA (*Clinical Features, Expectoration*).

Prunes.—*Prunum* is the dried ripe fruit of *Prunus domestica*, the plum. Prunes are mild laxatives, and may be added to the diet in cases of chronic constipation; they are contained in the *Confectio Sennæ* of the B.P. The *Prunus Virginiana*, or Wild Cherry, of the U.S. Pharmacopœia contains hydrocyanic acid, tannic acid, a volatile oil, etc., and is used as a tonic and sedative in diseases of the stomach and in coughs.

Prurigo and Pruritus.

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HISTORICAL NOTE.—In the days of Willan, and long after, the word Prurigo was a collective term for different itching affections of the skin. In those primitive stages of pathological knowledge classification could only be made by reference to superficial and obvious features. Thus any disease with an eruption of papules, accompanied by an intolerable sense of itching, was regarded as lichen, or strophulus, or as one of the prurigos. With fine insight Willan recognised that pruritus was the "leading circumstance" in the pruriginous affections. These he arranged in groups which, in accordance with the fashion of the times, he called "species," taking for his distinguishing mark such obvious features as degree of severity, extent of the disease, and the period of life. His so-called species were P. mitis, P. formicans, P. senilis, P. localis; and he placed them all in the order of Papulæ. The views of Willan prevailed in England, France, and Germany until Hebra dismembered the order. Hebra admitted as prurigo what Willan named P. mitis and formicans, and he maintained that the other eruptions were simply the result of itching of the skin, or pruritus. In the Hebra period classification had begun to feel the influence of the new science of pathology. The internal forms of eruptions were being revealed for the first time. Thus, in respect of the lesion of prurigo, what to Willan had been merely a papule became to Hebra an intra-epidermic cyst, which he regarded as a specific lesion, and the actual cause of the patient's suffering. His extraordinary experience of this disease, the regularity with which the symptoms grouped themselves in a striking clinical picture, convinced Hebra that this disease was to be regarded as an "entity," and to be entirely separated from the other pruritic affections. The force of Hebra's opinion carried the day, at least in England and Germany. Prurigo became an independent substantive disease, and pruritus was defined as itching *sine materia*, and itching affections provoked by epizoa were named after the parasite which was present. This is the current opinion in Great Britain at the present time, though Mr. Hutchinson has strongly protested against it. It has the merit of being easy of comprehension and is free from mystery.

The third period, through which we are now passing, may be styled the philosophical, and is dominated by the French writers. To attempt to give even a sketch of the opinions and counter-opinions of Vidal, Tenneson, Tommasoli, Brocq, Besnier, Foulard, Jacquet, Neisser, and others would demand many pages. But the volume of controversial literature is evidence that the old hard and fast lines of definition of the Hebra period are breaking down. Modern pathology is half a century in advance of the days of Rokitansky. Now we see the folly of impounding diseases within fixed circles of definition, and the tendency of the scientific nosologist to-day is to look past the superficial and obvious features of diseases for deeper and deeper resemblances having relation to their origin. From this results the modern arrangement of diseases into natural *series*. Thus the French writers, in endeavouring to establish a natural prurigo series, are quite in touch with the spirit of the times. The growth of our clinical knowledge, they say, has brought to light many cases of atypical varieties of prurigo Hebræ, and cases which have very little superficial resemblance to prurigo, but which present points of deeper resemblance. They maintain that the prurigo of Hebra is not an entity, but a symptom group which stands out in bold relief amid other groups less regular in form, and of lower

degrees of constancy. Those who take this view regard the eruption on the skin in all varieties of prurigo and pruritus as merely reactions of the integument to traumatism, or to accidental microbic infection. This has been strongly insisted upon by Besnier and Brocq in France, by Hutchinson in England, and by Touton of Wiesbaden. On the other hand, Tommasoli regards the firm sero-papule, or, as we should say, the cystic papule, as the essential lesion of prurigo. For this reason he excludes from the prurigo series the lichenified forms of Brocq. But whoever holds this opinion is logically bound to accept the inference, namely, that the cystic papule is antecedent to the pruritus, and, in short, the actual cause of the disease and of the patient's sufferings. Further, if the lesion is specific its formation should follow a definite routine, and the microscopists would, in the main, agree as to its form and character. This, however, is not the case. Scarcely two histologists agree in their results (*see* Anatomy of Prurigo, p. 329). The cystic papule of prurigo is frequently found in eczema. Again, cases of prurigo occur, as shown by Neisser, in which prurigo begins with urticarial lesions and not with the sero-papule. Jacquet has proved experimentally that pruriginous lesions may completely disappear simply by excluding them from all exterior influences, while at the same time the pruritus persists. In the light of this evidence we can only conclude with Besnier and Brocq that the eruptions on the skin in prurigo and allied disorders do not form an essential part of the disease, but are merely the reaction of the integument to traumatic influences. Our knowledge of these auto-traumatic lesions, or traumatides as I have ventured to call them (*see* p. 327), has been advanced by Brocq, who in 1891 lectured on the "theory of lichenification" (p. 327).

Beyond this point we enter on more debatable ground. Whether we should follow Besnier's arrangement into four groups, each labelled *Prurigo diathésique*, and differing according to the objective mode of cutaneous reaction, or Brocq's four varieties of *Névrodermite*, is a matter for the future to decide. In the present article I have treated *Prurigo Hebræ* as a symptom group. I recognise the existence of other groups belonging to the same series. But the arrangement adopted is categorical rather than scientific.

General Characters of the Prurigo Series

Before any disease can be admitted into the prurigo series it is essential that it possess the following characters:—The affection of the skin must first be latent. Without giving any eruptive sign the skin of the individual falls into a certain state, of which the first symptom is a sensation of itching or pruritus, this being followed by moderate, or immoderate, bodily excitement (scratching), which provokes traumatic lesions in the skin.

Pruritus.—To the question, What is pruritus? the physiologists of to-day can give no certain answer. Indeed, what little information we possess is the result of introspection based on a few facts culled from common experience. In order to awaken this sensation we only need to touch the surface with the point of some delicate object, such as a hair or the barb of a feather. Physiologists have elicited the fact that this property is not equally distributed all over the surface. Thus, if the back of the hand be touched lightly with the point of a hair, scarcely any sensation of itching is provoked, but if the side of the nose be touched in like manner there instantly arises a strong sense of itching. And, what is very curious, this sensation persists for an appreciable interval of time after the irritating object has been withdrawn. The sensation is rapidly followed by a considerable amount of bodily excitement. The magnitude of this excitement is out of all proportion to the delicate and feeble stimulus which provoked it. The excitement runs to a crisis commonly called scratching. After this there is a sudden abatement of the sensation.

Thus the word pruritus connotes not merely a sensation, but a series of processes beginning in some latent changes in the surface of the skin and ending in scratching. These are not of necessity morbid processes. Itching is a property of the skin, and is to be regarded as one of the common sensations peculiar to the integument of all warm-blooded animals. As Professor Bronson points out in his admirable essay on the *Sensation of Itching*, pruritus is closely associated with the animal sense of contact. He affirms that the sensations originating in the surface of the skin, that is, in the epidermis, are of a low order. They fail to convey to the mind any definite perceptive ideas regarding the properties of the thing that touches the surface.

This is evidence that the sensation of itching starts in the undifferentiated nerves of the epidermis, and proceeds to the spinal cord without throwing into action the deeper specialised sense organs of the skin. No fact of common observation or in cutaneous pathology contravenes this conclusion, so we may take it for granted that *the seat of pruritis is the epidermis.*

Physiology has not yet given us a satisfactory reason for the muscular excitement which follows the pruritic stimulation of the free nerve ends. Bronson and Payne explain it by the vagueness of the stimulation and the involuntary desire to substitute "an effective energy for an ineffective vexation" (Bronson). It cannot be denied that the act of scratching is *apparently* directed to a certain end, and so

far we can say that a vague conception is usually associated with pruritus. But when a man who is asleep, or even comatose, tears at his skin, it is obvious that neither conceptions and desires nor any other psychological act have anything to do with his pruritus.

To pursue this question further would only lead on into metaphysics. It is wiser to wait until physiology furnishes us with some definite facts.

The Causation of the Pruritic State

It cannot be insisted on too much that pruritus is the symptom and not the disease. The real disease underlying all the prurigos is latent. Of itself it produces no lesion; it rouses no cellular reaction, and no inflammation. Our ignorance of it is profound, therefore we write of it in terms of wide generality and often of vague and inexact meaning. It begins insidiously, and grows with the individual; often it is the only symptom, and the patients make no complaint of ill-health. The bodily functions, so far as we can detect, may be in no way disturbed. It sometimes occurs along with other symptoms, forming what we may call indefinite cases. Again, it may be associated with pronounced diseases of certain organs, for example, obstructive jaundice. It may assert itself in gout, in uræmia, and in Graves' disease. I have known it to be the most distressing symptom in general lymphadenoma. No period of life is exempt from it. In that particular manifestation of the pruritic state which we call prurigo Hebræ it begins in infancy or early childhood. It certainly assumes new aspects at different ages. The form of an adult prurigo is generally different from that of the child, while often in old age the pruritic state is lesionless. Some varieties are met with only among the very poor, but the well fed are not exempt. Of temperaments it may be said that the neurotic are prone to it, but some bad forms are associated with what we call the strumous, and the French the lymphatic temperament. But out of all this uncertainty we can at least fix this general truth, namely, that a *pruritic state* springs from antecedents which do not follow any routine track. Like fever, it is a perturbation of wide generality from which no warm-blooded animal is exempt.

According to Besnier, the main pathological condition of the prurigos lies in the adulteration of the blood, or of the lymph, or of the intercellular fluids. This adulteration he believes to be autonomous. That the stimulus which provokes the sensation of itching is chemical in its nature receives the support of the eminent physiologist Professor Kiesow.[1]

The Consequences of the Pruritic State; Traumatides.—For convenience of description we may divide these into (1) Cutaneous, and (2) General, or those which relate to the health of the individual.

1. Cutaneous.—To save repetition, and for brevity's sake, I propose to speak collectively of these scratch lesions of the skin as *traumatides.*

It is the merit of the modern French school of dermatologists that they have insisted on the fact that the essential disease of the prurigos is *latent.* By this is meant that the skin of an individual may gradually fall into certain pathological states of nutrition, and at the same time give no objective sign of it until provoked to do so by scratching. Then a variety of lesions arise. According to Besnier and Brocq, the form of the lesion is not controlled by the scratching, but by the individual himself. Traumatides are in themselves banal, and possess no fixed anatomical character. Their importance is derived from the fact that *they indicate the state of nutrition of the skin at the time of the traumatism.* The following traumatides have been observed:—

(1) Wheals.—Urticarial lesions may be regarded as traumatides. They occur most readily in infants and children, and indicate a state of tension in the vaso-muscles (probably veins), which when stimulated contract spasmodically.

(2) *Scaly spots;* these are due to the loosening of the horny epidermis.

(3) *Excoriated red spots* are formed by tearing fragments of the hard epidermis from the underlying soft epidermis.

(4) Vesico-papules or cystic papules.—These are common in childhood, adolescence, and in adults, but not so frequent in the aged. They are due to the forming of cystic cavities, with fluid contents, in the epithelium.

(5) *Broad hemispherical small nodules*, pale at first, but later becoming red. These are characteristic of prurigo Hebræ. They are due to the formation of cystic cavities, but situated deeper beneath the horny layer.

(6) Torn papules bearing at their summits a minute blood scab.—These are the commonest of all traumatides, and are met with in persons of all ages. They are a variety of the cystic papule.

(7) Pustules, superficial abscesses, and boils.—These are the consequence of microbic infection. They are most readily formed in persons of strumous temperament. Individuals of this class are distinguished by thick, coarse skin, with large sebaceous follicles. The lips are usually thick ("lymphatic") and the eyelids thickened.

(8) "Lichenification."[1]—Brocq was the first to give us, in 1891, an exact description of this form of traumatide. "When," says this

[1] This opinion was conveyed in a letter from Professor Kiesow to the writer.

[1] Besnier prefers the term lichenisation. Primary lichenification of Brocq-Jacquet corresponds to Vidal's lichen simplex chronicus circumscriptus, a term which, even in France, will soon be obsolete.

authority, "a person who is predisposed, and the predisposition is indispensable, repeatedly scratches some circumscribed patch of skin which is the seat of pruritus, it may determine more or less quickly certain morbid changes. The skin thickens, becomes hard and rugose, the papillæ hypertrophy, and groups of hypertrophied papillæ produce papules of irregular and unequal size. The normal folds and surface markings may be exaggerated, forming a sort of quadrilateral mesh. The integument is more or less infiltrated, and has lost its suppleness and normal consistence." To this alteration of skin Brocq gave the name "lichenification." When this lichenification arises in a skin apparently healthy it is termed primary lichenification. Far more frequently it is secondary to some previous eruption, such as eczema or lichen planus.

The rapidity with which, in some cases, large islands of pigmented and indurated, or "lichenified," tissue may arise in a circumscribed area, which is the seat of pruritus, without any previous signs of abnormality, is one of the strongest proofs that the nutritional state of the skin may deviate considerably from the normal without giving any spontaneous eruptive symptom. This form of traumatide is met with in neurotic persons, and may be associated with jaundice. It sometimes follows mental shock or states of mental worry and depression.

(9) Eczematisation.—Besnier has applied this term to patches of superficial inflammation presenting all the polymorphic characters of eczema, namely, hyperæmia, vesication, oozing, desquamation, and suppuration.

(10) Pigmentation.

(11) General dryness of the surface.

General Consequences of the Pruritic State

If we are to judge of the importance of a pathological condition by the greatness of the consequences which proceed from it, there are few more important than those of which pruritus is the symptom. If the itching is severe the sense of torment replaces every other consideration. To work is impossible, and to sleep no less so. The power of concentration is weakened, and with loss of control there is developed an irritable temper. The unfortunate person falls into a "nervous condition." The approach of night brings no promise of relief, for sleep, which subdues pain, has no power to arrest pruritus. The disturbance in the peripheral nerves and the spinal excitement still goes on. Too often the patient is sleepless, and makes frantic efforts to relieve his torment by tearing at the skin. Should this continue without intermission it would speedily end in madness, but the irritation is intermittent and paroxysmal, and in the calm intervals he recovers to some extent. If the pruritus is acute and temporary, or even if it is chronic, provided it is not of a severe character, the general state of nutrition may even be good (Hebra). But in many cases the entire nutrition of the body suffers, and this is generally observed in neurotic adults. The misery of these unfortunate persons is increased by their social ostracism. Pain wins sympathy, but pruritus only aversion and annoyance. In childhood he is scolded by his teachers and jeered at by his school-fellows, and later in life he comes to be avoided as an object of contagion, since the act of scratching is always suggestive of parasites. Thus the whole life of the sufferer is dominated by his complaint, his lot is isolation, and we need not wonder if it drifts into melancholia, or terminates in suicide.

Clinical Groups in the Prurigo Series

In sketching the general character of the prurigo series it was pointed out that the essential disease underlying all forms of prurigo and pruritus is some latent change in the nutrition of the skin, which is accompanied by an intermittent and paroxysmal sensation of itching, and later by the appearance of traumatides or scratch lesions. The present imperfect state of our knowledge of the pathology of the pruritic states forbids us even to attempt a scientific classification of these disorders. All we can do is to arrange our cases into groups, according to circumstances which seem to us more or less striking. The result of the arrangement is merely a catalogue of cases, and is not to be regarded as a natural classification.

Prurigo Hebræ.—*Pruritus in badly nourished Children, followed by pronounced Malnutrition of the Skin and severe Traumatides, continuing more or less through Life.*—According to Hebra, the disease begins in infancy and continues throughout life. The earliest objective symptom is that of subepidermic papules, which form in all parts of the body surface, except in the skin covering the bends of the joints. The face, throat, and neck are rarely affected to any extent. The papules are isolated, and increase in number in the lower limbs. In long-continued cases the whole nutrition of the skin suffers. It becomes harsh and rough to the touch, of thicker consistence and of darker complexion than in health. The itching is more or less constant, but subject to exacerbation and paroxysm, especially at night. Hebra firmly maintained that the subepidermic papule was the essential part of the disease, and that it was the inevitable antecedent of the itching. This, by many, is considered doubtful, and the evidence points rather to the lesion being secondary to the scratching. In consequence of scratching, the skin over the papules becomes reddened and excoriated, and the papules discharge a fluid which dries into a scab. If the lesions below the knee are numerous the inguinal glands inflame and become enlarged, forming the so-called prurigo bubo. When the disease is fully established the general aspect of

the patient is highly characteristic. When he is stripped for examination we notice that there is a clustering of the lesions chiefly below the hips, the maximum development being reached below the knees. As the disease ascends it becomes feebler, and the lesions more widely scattered, till they cease on the neck and face. The general type of nutrition is commonly strumous or "lymphatic." The lesion most in evidence is the torn, scabbed, broad, hemispherical papule, often intermixed with ecthymatous pustules; in some cases with patches of infiltrated eczema.

Etiology.—The causation of prurigo is unknown. We know of no irritant which can produce it. It commonly appears during infancy, but, according to Neisser and Ehlers, it may originate later in life. According to the latter of these authorities the date of onset varies from a few days to twenty-nine years of age, the average of 207 cases being five and a half years. Hebra maintained that prurigo was never congenital; Ehlers, however, believes heredity plays an important part in its causation. "It occurs," says Hebra, "almost exclusively in poor subjects and those ill-nourished in childhood, and so most often in foundlings and beggars' children. Those who have enjoyed a good physical education in early youth, and have always been properly fed according to their age, suffer very rarely indeed from prurigo." The prurigo of Hebra has curious geographical relations. In point of frequency Vienna seems to be the headquarters of the disease, and this city is the centre of a mixed population of Austrians, Germans, Tyrolese, Slavonians, Hungarians, Jews, Italians, etc. It has been noticed that the offspring of sufferers emigrating from Vienna to a country where the conditions of life are easier and more wholesome do but rarely manifest the particular group of symptoms which have been described above. Professor James C. White, of Boston (U.S.A.), has made a careful study of the non-prevalent distribution of "prurigo" in the United States. It appears that prurigo is an almost unknown disease among the American people as a whole, and it is only rarely observed in immigrants from European countries and in their descendants.

Anatomy.—Hebra and Simon regarded the elementary lesion of prurigo as a species of vesicle. Kaposi regards it as analogous to the papule of eczema. According to Riehl, they are similar to the papules of urticaria. Vidal considered them to be analogous to the papules of lichen. Wedl and Neumann believe that they are the product of exudation into the cutis. Auspitz and Caspary consider that they are the result of proliferation of the rete Malpighii. Leloir found the prurigo nodule to be associated with a cavity formation in the prickle layer. According to this authority, the cysts are formed by the destruction of prickle cells, and not by exudation. The cysts enlarge, but never invade the horny layer. Kromayer found minute cysts in the horny layer, and accompanied by hypertrophy of the epidermis, with some cell infiltration in the upper part of the cutis. Epidermic cysts have also been noted by Campana and Bonfigli. Prurigo papules a few hours old have been examined by Darier. He found the lesions to consist of an area of colloid degeneration in the epidermis surrounded by signs of acute inflammation, namely, œdema, vesication, and leucocytosis. According to Du Mesnil, the primary anatomical change is the thickening of the epidermis, which leads to the retention of sweat in the sudoriparous ducts, and so to the formation of cysts. These varied and contradictory opinions are of themselves quite sufficient proof that the lesions of prurigo are banal, and have no definite anatomical character.

Diagnosis.—Prurigo Hebræ being a rounded and complete symptom group, diagnosis depends on our acquiring a general impression of the disease rather than upon the detection of minute details. The diseases with which it may be confounded are those in which itching is a leading feature. In every supposed case an examination for parasites should be made, for the presence of epizoa alters both the prognosis and the treatment. The distinction between pruritus and prurigo, in the broadest sense of the term, is perhaps more academic than scientific. The essential basis of every case of prurigo is pruritus, that is, a tormenting sense of itching. For convenience' sake we separate the terms, and employ the name "prurigo" where the subjective and objective symptoms are grouped in a certain way.

Scabies is distinguished from prurigo by the detection of burrows, or the acari, and also by the history and nature and grouping of the lesions. *Chronic eczema* is not accompanied by the degeneration in the nutrition of the integument, nor by the scattered, torn hemispherical papules which are characteristic of prurigo. In chronic *pediculosis* the lesions are chiefly on the back between the shoulders, and the pigmentation of the skin is usually marked. The examination of the clothes will generally decide the question.

Prognosis.—1. Hebra regarded the disease as hopelessly inevitable. Ehlers found improvement after puberty. It seems certain that unless the circumstances of the patient can be greatly improved no hope can be looked for. The disease, however, is subject to remission. During the summer months Hebra noted a considerable amelioration of the symptoms, the disease being always worse in winter.

2. *Pruritus followed by urticarial traumatides occurring at all periods of life, but more frequently in infants and children.*—This will be discussed

under the head of Urticaria, but I believe that the proper nosological place of this class of disorder is in the prurigo series.

3. *Pruritus beginning in adult life and followed by traumatides of the nature of torn papules showing no tendency to aggregate by fusion, and scattered discretely, especially over the extensor surface of the limbs.* (*Prurigo simplex*, Brocq. *P. temporaria*, Tommasoli.)

(1) *Acute Form.*—According to Brocq, the onset is rather sudden, but without constitutional disturbance. The eruption takes the form of firm, solid, red papules, often torn at the summits and tipped with a minute scab. The skin is in the urticarial condition, and wheals may be present. The papules, which are always discrete and never fused into patches, are distributed chiefly over the extensor surfaces of the limbs, especially the forearms, elbows, knees, and dorsal surfaces of hands and fingers. The flexor surfaces of the limbs and the bends of the big joints are free, or only very slightly affected. The disease runs a course of from two to eight weeks, and ends in recovery. Relapses are frequent. Lichenification and eczematisation are not met with in this variety. According to Darier, the papules of P. simplex have the same anatomical character as the papules of prurigo of Hebra. This variety is practically identical with lichen urticatus of children.

(2) *Chronic Form.*—Objectively indistinguishable from the acute form, but continuing for months or years. Both these varieties are apt to occur in highly neurotic persons.

4. *Pruritus followed by traumatides secondary to mental shock.*—Many cases are now recorded in which a sudden mental shock or great grief has been followed by pruritus. The traumatides are not always of the same character. In some cases the lesions take a distinctly lichen planus form. In some of these cases the pruritus may be local instead of general.

5. *Pruritus with traumatides recurring in successive winters* (*P. hiemalis*, Duhring).—This class of case, described by Professor Duhring, of Philadelphia, under the name of *Pruritus hiemalis*, is characterised by the fact that the pruritus occurs only in the cold weather. As a rule it appears between October and January, and disappears in April or May. The whole body may be affected, but generally it is worse on the extensor surfaces of the limbs, especially on the thighs. The itching is worse on going to bed and on rising in the morning, and less troublesome in the middle of the day. The traumatides are not pronounced in character, but the skin may become rough, excoriated, and marked by scattered, torn papules. Persons who suffer from this form of pruritus have sometimes remarkably dry skins. According to Corlett, the state of the general health has no appreciable effect on the pruritus. Local irritation of clothes may aggravate, but does not produce it. He believes that the meteorological conditions most apt to provoke the pruritus are a low temperature, low humidity, with N.W. winds. The greater the velocity of wind the greater the pruritus.

6. *Pruritus followed by traumatides recurring in successive summers.*—These cases are rarer, but individuals exist who are more sensitive to heat than to cold, and whose pruritus disappears in the winter to reappear in the summer.

7. *Pruritus following pregnancy and menstruation* may be local or general. In certain women it occurs with each pregnancy and subsides after delivery. There is nothing to distinguish it from other forms of pruritus except its association with the physiological events of pregnancy and menstruation.

8. *Pruritus with pronounced and multiform traumatides concurrent with jaundice, uræmia, diabetes, or intestinal bacterial toxæmia.*—According to Besnier, the immediate irritant is not the bile pigments, urea, or sugar, but some irritant liberated by these agents. There is some condition of which we are ignorant, for there are many cases of jaundice, uræmia, and diabetes in which no itching occurs. Objectively this group is characterised by traumatides of the nature of lichenification and eczematisation.

9. *Pruritus without traumatides in the aged* (*P. senilis*).—These cases are characterised by the fact that scratching does not produce traumatides, or at most only a few slight lesions. It is supposed to be due to atrophic degeneration of the skin. In some cases it is associated with lithæmia, obstructive jaundice, chronic Bright's disease, and diabetes mellitus. When pruritus becomes established in old people it is incurable.

10. *Pruritus affecting restricted areas* (*P. localis*).—It is a good rule to assume that all local forms of pruritus are due to local causes until *proved* not to be so.

(i.) *P. vulvæ in children* is often reflected from the rectum, due to the irritation of ascarides.

(ii.) *P. vulvæ in adults* may be provoked by saccharine urine, or acrid vaginal discharges. It may be a premonitory symptom of carcinoma uteri (Kaposi). In many cases we entirely fail to discover any cause. Some persons have a nervous history. One of my patients presented the following family history: two brothers were insane, the father was "peculiar," and had been afflicted with sexual incompetency; the paternal grandfather was at one time insane; the mother, now living, is highly neurotic, and would be described in society as an "oddity." The itching affects the labiæ and the clitoris, and often the lower part of the vagina. Prolonged scratching is apt to lead to great induration of the parts, and to the formation of papules. But in other cases traumatides may be absent, or

the labiæ may be slightly hyperæmic and excoriated. Pruritus in this region is apt to produce depression of spirits. According to Bronson, it may be associated with voluptuous sensations.

Pruritus ani.—The pathological conditions which give rise to this troublesome affection are commonly to be found within the sphincter or in the lower third of the rectum. If the anus is dilated, minute fissures may often be seen lying between the folds of skin. Slowness of circulation, especially if combined with varix or hæmorrhoids, is a frequent cause of pruritus. In certain cases some article of diet, such as coffee, occasions the itching. Itching in this situation is intolerable, and scratching leads to eczema, with formation of fissures. Gouty and neurotic persons are more prone to it than others. In women it may appear at the menopause. It must not be forgotten that anal pruritus, like that of the vulva, may be a symptom of pathological changes in the uterus, such as fibroma or carcinoma, or of carcinoma of the rectum.

Pruritus palmæ et plantæ is very rare. Gouty persons are more prone to it than others.

Pruritus linguæ has been recorded in one or two instances.

Treatment

I. Management of the Individual. — (1) *General measures referable to all cases of pruritus, whether associated with prurigo lesions or not.*— *Food* must be of good quality, and suitable to the age of the patient. The relative proportions of fats, carbohydrates, and proteids have not yet been determined, but every case must be studied on its own merits. Ivanow recommends the introduction of a large quantity of fatty food into the dietary of prurigo children. A meal rich in proteids is apt to aggravate the sensation of pruritus. All forms of alcohol are calculated to increase cutaneous irritation, especially champagne and liqueurs, but in some cases in which nutrition is subnormal and feeble whisky and water or dry wines may be advisable. Brown-Sequard draws attention to the action of coffee provoking or aggravating pruritus. If atony or dilatation of the stomach exists, the meals should be light and should be taken at shorter intervals.

Air.—This should be abundant, and as pure as possible. If the humidity is too low the air may cause irritation. Wind, to some individuals, is a direct cause of pruritus.

Light increases metabolism, and so far may affect the pruritus. Its physiological action must also be borne in mind. *Fermentation* and *catarrh* in any part of the alimentary canal must be corrected. If *muscular atony* and dilatation of the stomach exist, massage and graduated exercises will prove beneficial.

(2) *Special measures to be selected according to the nature of the case.*—In adults suffering from pruritus or prurigo, *rest* is the great desideratum. By this is not meant idleness. It is highly desirable to enliven the patient's interest in things around him. What is needed is a true adjustment of the individual to his work, that this be so ordered that every part of it becomes effective. Nothing is more calculated to produce depression and to wear a man down than ill-directed work ending in failure. Harmonious work is a great therapeutic agent. Many a patient may be saved from misery, if not from insanity, by giving him a better method of work. And this principle applies to the holiday times no less than to the hours of business.

Climates for pruritus.—This is one of the most valuable of measures for restoring the pruritic patient, especially if associated with exhaustion from overwork, or with functional disturbance of the nervous system. But whether we send him far or near, to sea or country, to hill or valley, must be determined by a variety of circumstances which calls for wise discrimination. Brightness and pure air are good, but there must be sympathy between the individual and his surroundings. To send on a long voyage a hypochondriacal person who hates the sea is to invite him to commit suicide. If such an individual were something of an antiquarian, he would have a better chance in the British Museum studying coins or mummies. If there is much exhaustion, or feebleness of constitution, a long journey is to be avoided, nor should the climate be too vigorous. High altitudes are not favourable to hysterical natures, but are highly beneficial to the lymphatic and sanguine. On the whole, a warm, equable climate is the best—one in which the indoor temperature corresponds with that out of doors. Countries of sharp thermal contrasts, as between hot interiors and freezing exteriors, and between middays and evenings, are to be avoided by the pruritic. This is probably one of the reasons why pruritus is so prevalent in the north-western States of America, where the people are exposed to great disparity of temperature.

Insomnia is one of the great difficulties with the pruritic. Brain sedatives, Dr. Payne thinks, are advisable. He recommends chloral in full doses, and antipyrin. Bulkley speaks well of gelsemium and cannabis indica. A complete hot bath, especially if bran or starch be added to it, and taken at bedtime, sometimes affords great relief. Brocq strongly advocates the daily hot douche. Electrical treatment has proved serviceable in some cases. The well-to-do may avail themselves of the electric bath, or large sponge electrodes connected with an induction coil may be moved over the surface of the trunk and limbs.

II. The Management of the Skin.—When, in the course of pruritus, the nutrition of the

skin becomes affected, measures are required to restore it to a healthier state. The hot bran bath followed by inunction can do much towards this. Harsh dry skins should always be anointed with oil or salves morning and evening. The addition of tar to the ointment has a sedative effect. Kaposi recommends beta naphthol, and more recently epicarin, one of the naphthalin compounds. If there is much infiltration and thickening of the skin Hebra's soap treatment should be tried. This consists in washing the skin with a flannel and the spirit solution of soft soap, afterwards applying some soothing ointment. If the pruritus is confined to a limb, the occlusion method recommended by Jacquet and Tenneson may be tried. Jacquet's plan is to fold cotton-wool round the limb, and apply over it a bandage in such a way as to exclude air and traumatism. The results are described as astonishing; all the lesions disappear, and in some cases, but not all, the pruritus also. Tenneson advises the use of guttapercha tissue or indiarubber dressings. Colombini speaks favourably of menthol dissolved in spirit. Cocain (20 per cent) applied locally in the form of ointment or lotion gives temporary relief, but does nothing towards a cure. Hatschek reports favourably of massage. Pilocarpine injections are recommended by R. M. Simon. Good results are said to have been obtained by the continuous electric current when the pruritus is confined to a limited area. But, after all, none of these measures touches the root of the disease. The only rational treatment is that which is based on a study of the individual himself.

Treatment of local pruritus. — Only two varieties need be discussed under this heading, namely, P. vulvæ and P. ani.

P. vulvæ.—All local sources of irritation must be searched for and removed as far as possible, such as vaginal discharges, fissures, saccharine urine, ascarides, hæmorrhoids, and morbid conditions of the uterus. Care must be taken not to overlook the presence of parasites. If eczema be present, this must be cured (*v.* art. "Eczema"). If the labiæ are thickened, carbolic or tar ointment may be applied. In some cases the cautious application to small areas of 95 per cent carbolic acid gives relief, or even removes the pruritus. In obstinate cases, burning the parts thoroughly with the thermo-cautery is effective, and may result in a complete cure. For this operation general anæsthesia is necessary, and the patient will require to remain in bed for three or four weeks.

P. ani.—The treatment of this is similar to that of P. vulvæ. We must remember that it more often depends on defective circulation, and that gouty remedies may be necessary. The patient's diet may require regulating. The abuse of coffee should be stopped. As regards local treatment, Ohmann-Dumesnil recommends the application of pure creasote. Phenol may be used as for P. vulvæ, but neither this nor the creasote should be applied on consecutive days. If the anus be kept firm and dry it will add much to the comfort of the patient. This may be attained by sponging the parts with a 0·5 per cent solution of silver nitrate or acetate of lead in spirit, water, or nitrous ether, and after this dusting *into* the anal passage a drying antiseptic powder.

Pruritus. *See* PRURIGO.

Prussak's Space. *See* EAR, ACUTE INFLAMMATION OF MIDDLE EAR (*Introductory, Tympanum*).

Prussian Blue and Prussiates. *See* POTASH AND POTASSIUM.

Prussic Acid. *See* HYDROCYANIC ACID; TOXICOLOGY (*Cyanogen Compounds*).

P.S.A. — These letters, in Public Health, stand for Port Sanitary Authority, which has to do with the hygiene of ships as to overcrowding, infectious diseases, etc.

Psalterium.—A thin lamina of fibres in connection with the fornix and corpus callosum of the brain; the lyra; also the omasum or manyplies (*q.v.*).

Psammoma. *See* BRAIN, TUMOURS OF (*Morbid Anatomy*).—It is a tumour (fibro-sarcomatous) found in connection with the brain, and containing calcareous concretions.

Psellismus.—Defective articulation (Gr. ψελλός, unable to pronounce certain syllables).

Pseud- and Pseudo-.—In compound words *pseud-* and *pseudo-* (Gr. ψευδής, lying or false) mean false or spurious; *e.g. pseudarthrosis* (false joint), *pseudacusis* (disturbed sense of hearing, in which the individual hears voices falsely as to timbre and pitch), and *pseudaconitine* (false aconitine).

Pseudæsthesis.—The imaginary feeling of the presence of organs or parts which have been removed, *e.g.* the sensation of moving the toes after amputation of the leg.

Pseudechis. — *See* SNAKE-BITES (*Colubrine Snakes, Elapidæ*).

Pseudoanæmia.—Apparent anæmia, as in Bright's disease, arteriosclerosis, lead-poisoning, nausea, etc.

Pseudo-angina.—False, spurious, or hysterical angina pectoris. *See* ANGINA PECTORIS (*Varieties*).

Pseudoapoplexy.—Peculiar seizures,

resembling apoplexy, which may occur in cases of fatty degeneration of the heart.

Pseudoappendicitis.—A hysterical affection simulating inflammation of the appendix vermiformis.

Pseudobacillus.—This term is given to fat crystals staining like the tubercle bacillus and occurring sometimes in sputum.

Pseudobiliary Colic.—Acute pain in the upper part of the abdomen resembling that met with in cases of gall-stones, but due entirely to nervous causes.

Pseudoblepsis. — A hallucination of vision.

Pseudobulbar Paralysis.—Bulbar paralysis of *cerebral* origin. *See* Brain, Affections of Blood-Vessels (*Paralysis, Pseudobulbar*).

Pseudocavernous Signs.—The physical signs usually associated with the presence of a vomica in the lung, but due in this case to the contiguity of an area of consolidation to a large bronchus.

Pseudochromæsthesia.—An "anomaly in the perception of visual sensations, in which the vowels in words appear coloured, each having a different tint" (Hack Tuke).

Pseudocroup.—False croup; laryngismus stridulus, due, for instance, to post-nasal adenoids. *See* Nose, Post-nasal Adenoid Growths (*Symptoms, Nervous*).

Pseudocyesis, or Spurious Pregnancy. *See also* Climacteric Insanity (*Pseudocyesis*); Hysteria (*Digestive Organs, Disorders, Tympanitis*); Menstruation and its Disorders (*Amenorrhœa from Nervous Disturbance*). —This comparatively rare condition is a variety of a group of cases recognised under the heading of "Phantom Tumours." It occurs chiefly amongst those who have recently been married, or who are approaching or have reached the menopause, or in cases where illicit intercourse has occurred followed by fear of pregnancy. Again, in some cases no reason can be assigned for the occurrence of this phenomenon, while in others it may arise as a result of an eczematous patch in the neighbourhood of the umbilicus, as in a case described by Sir William Bennett, or as a result of pelvic peritonitis, which produces partial obstruction of the sigmoid owing to the formation of adhesions. Opinions differ as to method of formation of such tumours. Some distinguished authorities, amongst whom may be mentioned Addison, Gull, Paget, Spencer Wells, Bennett, believe that they arise by contraction of the abdominal muscles. While others, following the researches of M'Ardle and Kolipinski, hold that they are generated by an atonic condition of the intestines, which results in the accumulation of gas and consequently distension. Sir James Y. Simpson was of opinion that spasm of the diaphragm was the cause. It is worthy of note that such tumours are not confined to the human species; they also occur in the bitch and ass. Two types may be described amongst women who suffer from this condition: in one there is abdominal enlargement, and no other symptoms present except those of imaginary fœtal movements due to flatulence; in the other there is also abdominal enlargement, and in addition many of the symptoms and some of the signs of an ordinary pregnancy. Amenorrhœa, morning sickness, mammary changes, may be present in the latter type.

The diagnosis of the condition is easy, provided that one is not deceived by the statements of the patient, and that a physical examination is made. Suspicion as to the true state of affairs may be aroused by the mental attitude and age of the patient as well as by incongruity in her statements; signs and symptoms not developing in correct order, or being absent when they should be present. On examination it may be found that increase in the size of the breast and abdomen is due to the deposition of fat. In nulliparæ no mammary secretion can be expressed. Even in cases where the abdomen is the size of a full-time pregnancy the umbilicus remains depressed, and no fresh purple striæ gravidarum are present. On percussion over the tumour the note may be dull, but not so absolutely dull as over a pregnant uterus. Auscultation will give negative results, and though the intestines are distended with gas, borborygmi in most cases are not audible. On vaginal examination the usual signs of pregnancy are absent. Diagnosis may be confirmed by placing the patient under the influence of an anæsthetic. When complete anæsthesia has been reached the tumour gradually disappears. Bennett recommends that the hand should be gently placed on the tumour from the time when administration of the anæsthetic is begun. During the earlier stages it will be found to increase a little in size and hardness. With the onset of complete anæsthesia a little quivering motion will be felt, and then the tumour will gradually melt away under the hand. They never disappear suddenly.

Prognosis.—In many cases, especially amongst recently married women, the tumour and all symptoms of pregnancy disappear when the true state of affairs has been revealed. In others, again, the condition ends only when a spurious labour has convinced the patient of the truth of the obstetrician's diagnosis. While in some, and more especially in nulliparous women who have reached the menopause, it may be impossible to dispel the delusion from which

they suffer; the symptoms may last for years, and spurious labour recur at intervals.

Treatment of the condition in most cases lies entirely in convincing the patient that she is suffering from a delusion. The exercise of much tact may be called for. At the same time an endeavour should be made to ascertain the existence of an underlying cause, such as pathological pelvic conditions, and if discovered this should be attended to. Adiposity, dyspepsia, and flatulent distension should receive appropriate treatment.

Pseudodiphtheria. *See* DIPHTHERIA (*Problems in Pathology, Attenuation of the Bacillus*).

Pseudoencephalus or Pseudencephalus.—That form of anencephalus in which the basis cranii is covered with a soft, spongy, vascular mass.

Pseudogensia.—A taste hallucination or false perception.

Pseudoglobulin. *See* PHYSIOLOGY, BLOOD AND LYMPH (*Plasm and Serum, Globulins*).

Pseudohermaphroditism. *See* HERMAPHRODITISM.

Pseudohydrophobia.—The imaginary hydrophobia of a nervous person who has been bitten by a dog not suffering from rabies. *See* RABIES (*Diagnosis*).

Pseudohypertrophic Paralysis. *See* MUSCLES, DISEASES OF (*Idiopathic Muscular Atrophy and Hypertrophy, Pseudohypertrophic Type*); DEFORMITIES (*Congenital Dislocation of Hip, Diagnosis*).

Pseudohypertrophy. *See* HYPERTROPHY (*Pseudohypertrophy*).

Pseudoileus.—A form of intestinal obstruction. *See* OVARIES, DISEASES OF (*Complications following Ovariotomy*).

Pseudoleukæmia.—Hodgkin's disease. *See* LYMPHATIC SYSTEM (*Lymphadenoma*); RICKETS (*Associated Conditions, Pseudoleukæmia Infantum*).

Pseudolipoma.—Localised œdema, especially in the supraclavicular regions in cases of myxœdema.

Pseudomembrane.—A false membrane, *e.g.* in diphtheria (*q.v.*).

Pseudomeningitis. *See* HYSTERIA IN CHILDHOOD (*Symptoms, Pseudomeningitis Hysterica*).

Pseudomnesia.—A false memory, the individual so affected "believing that he remembers facts that never existed" (Hack Tuke).

Pseudomucin. A substance resembling mucin, found especially in some ovarian cysts (*q.v.*).

Pseudoparalysis. *See* RICKETS (*Clinical Features, Muscles and Ligaments*); SCURVY, INFANTILE (*Diagnosis*).

Pseudopsia.—False vision.

Pseudoptosis. *See* OCULAR MUSCLES, AFFECTIONS OF (*Paralysis, Etiology, Nuclear Ptosis*).

Pseudosclerosis. *See* PARALYSIS (*Disseminated Sclerosis, Diagnosis*).

Pseudosmia.—False sense of smell.

Pseudosteomalacia.—The malady of the bones due to *late* rickets and therefore simulating osteomalacia or malacosteon. *See* LABOUR, PROLONGED (*Pelvic Deformities, Pseudo-osteomalacic Pelvis*).

Pseudotabes. *See* NERVES, MULTIPLE PERIPHERAL NEURITIS (*Symptoms, Ataxia or Pseudotabes*).

Pseudotuberculosis.—A disease resembling tuberculosis met with in animals, and due to protozoa and to bacteria other than the tubercle bacillus; it is rare in man.

Psilosis. *See* SPRUE.

Psittacosis.—A disease of birds (Gr. ψίτταϰος, a parrot) which is believed to cause an atypical pneumonia in the human subject.

Psoas Abscess. *See* KIDNEY, SURGICAL AFFECTIONS OF (*Renal Fistula*); SPINE, SURGICAL AFFECTIONS OF (*Spinal Caries, Complications, Psoas Abscess*).

Psoas Muscle. *See* SPINE, SURGICAL AFFECTIONS (*Injuries of Lumbar Plexus*); HIP-JOINT, DISEASES OF (*Diagnosis of Hip-Joint Disease, Lesions of Psoas Muscle*).

Psoitis.—Inflammation of the psoas muscles.

Psora. *See* SCABIES.

Psoriasis.

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See also Alopecia (*Associated Diseases*); Gout (*Irregular, Cutaneous System*); Hydropathy (*Uses, Affections of the Skin*); Leucocytosis (*Eosinophilic, in Psoriasis*); Nails, Affections of the (*In Psoriasis*); Rheumatism, Acute (*Cutaneous Manifestations*); Rheumatism, Rheumatoid Arthritis (*Psoriasis*); Rheumatism, Chronic (*Clinical Features*).

Synonyms: *Lepra* (Willan), *Alphos* (E. Wilson) are both obsolete. Fr. *Psoriasis*; Ger. *Psoriasis, Schuppenflecle.*

Definition.—A chronic and recurrent disease, characterised by dry, red inflammatory discs almost concealed by a crust of adherent silvery scales.

Clinical Features.—Psoriasis is one of the most common diseases of the skin in Northern Europe, where it constitutes from seven to eight per cent of all skin diseases; while in Southern Europe and America it only occurs in from two to three per cent. In private practice it is the second most common disease.

Most of the varieties created by older writers were artificial, and little more than convenient labels for different stages in the evolution and involution of the disease, their chief justification being that the eruption may stop at any of the stages for a considerable time, and even disappear without traversing the complete gamut of development and involution.

Psoriasis commences as a papule the size of a pin's head, slightly reddened and scaly from the first, though sometimes a slight scratch with the nail is required to demonstrate the scaly cap. This stage is P. punctata. It soon flattens and enlarges peripherally to a scaly spot (P. guttata), and this again to a disc which may develop to or stop short at any size from a threepenny to a crown piece (P. nummulata). Isolated patches are seldom larger than this, but by coalescence with other patches an extensive area may be covered (P. diffusa), the borders of which are gyrately outlined. In the retrogressive stage, involution commences in the centre, by a diminution in redness and scaliness going on to complete clearing away, leaving a circle with raised scaly border and normal or slightly pigmented centre in an isolated disc, or an irregular, gyrately outlined area with the border one-eighth to one-quarter inch wide in a compound patch. In a few cases these circinate and gyrate patterns are formed primarily from the eruptive elements having formed at and followed the distribution of the hair follicles, which constitutes what Willan called Lepra, a term now restricted to leprosy.

Reverting to the nummulate stage, which may be considered as the most typical condition, it may be observed that the lesion is a disc slightly elevated above and sharply defined from the surrounding healthy skin. All but the extreme edge, and sometimes even that, is covered with a white crust of adherent scales, their silvery hue being due to inclusion of minute air-bubbles in their substance. This crust is not easily attachable until involution sets in, but if removed forcibly the exposed base is seen to be of a red colour, with still brighter red points upon it which bleed readily, and are really the tops of exposed and enlarged papillæ of the cutis. The thickness of the crust varies greatly, and in extreme instances the scales are heaped up into a conical crust like rupia, hence the term P. rupioides. In almost all cases the crust is entirely composed of scales, but occasionally there is slight pustular exudation mixed with them, hence P. empyodes.

Itching is not a marked feature as a rule, but in gouty and alcoholic patients, and whenever there is marked hyperæmia, the irritation may be very pronounced, and the consequent scratching produces thickening of the patches, blood crusts, and excoriations.

The distribution of the eruption is an important feature in the general picture of the disease. Symmetrical in the main, there is a selective affinity for the extensor aspects of the limbs, especially about the elbows and knees, where the patches are apt to aggregate and coalesce even when scattered discretely in various sized discs over other parts of the body surface. No part of the body is absolutely exempt from the eruption, but the palms, and soles, and lower part of the face usually escape altogether, or are but slightly involved; but sometimes they are the first and the worst parts affected. As a rule, next in order to the limbs, the scalp and the back of the trunk are most frequently and extensively involved. When every region of the body is extensively invaded P. universalis is present, but there are always some areas of healthy skin.

In a well-marked, fully-developed case, in a general survey, the eruption may be seen in all stages of development at once, affecting a large portion of the body surface; but however long the eruption has been out, and the patches have grown and coalesced, there are always some areas left clear of eruption. In most cases, however, there are in the first attack very few patches, and these may be confined to one or two regions, such as the knees, elbows, or scalp, for a very long time, and then either gradually or rapidly extend. On the other hand, in some patients who have had previous attacks there may be an abundant outbreak from the first; the eruption comes out in crops at short intervals, and speedily involves every region of the body. Between these extremes there are all gradations.

Most cases run a very slow course, and whilst some, especially young subjects, may lose their eruption spontaneously for a time, in others there are remissions only, the disease lasting, unless vigorously treated, for many years.

Whether the eruption is removed by time or by treatment, recurrence is the rule, to which there are but few exceptions. And whilst in some patients the interval of freedom is only a matter of weeks or months, a few go for years without a fresh attack. The eruption leaves behind it a redness or slight staining at the site of eruption, but in some, especially when treated with arsenic, the pigmentation is marked and persistent. The converse, however, may occur; transitory achromia is often left after treatment by chrysarobin, but in rare cases it occurs persistently without apparent cause, and even scarring may be produced with slightly increased pigmentation round the patch.

Even when the eruption is very extensive the general health is unaffected by the skin lesions, but their condition is often influenced by the general health of the patient, deterioration in which may determine an outbreak or aggravate an attack.

Acutely inflammatory cases present a totally different picture from the subacute, more typical form previously described. While the extensor aspect of the limbs is still the most frequent position for it to start from, it is much more indiscriminate in its origin and distribution (in one case under the author it always began on the palms and soles), large areas are speedily involved, and the borders are much less defined than in the familiar discs; the ground colour is an intensely bright red, and the scales are not in glistening adherent crusts, but in flaky scales which shell off readily and frequently in large quantities, the general aspect approaching that of pityriasis rubra, into which it sometimes merges and then runs the course of that disease.

Limited areas with these characters may be seen sometimes in elderly persons, and even have a somewhat eczematous character though the patch is generally dry, and the condition Devergie called P. eczemateux is produced.

On the palms and soles, it is seldom seen without being present extensively elsewhere. It usually shows itself by thickening of the horny layer in small patches, and when this splits off it leaves whitish spots with a worm-eaten appearance. It is sometimes seen in scaly patches which may be heaped up considerably, but has not the lamellar aspect of the disease in other parts. In a few instances the disease has been limited to the hands, including the palms, for many years. The term palmar psoriasis is sometimes misapplied to dry palmar eczema and a secondary scaly syphilide of the palm.

Sweat-duct psoriasis is generally unrecognised. The lesions are only one-eighth of an inch in diameter, but may cover a great part of the trunk or limbs. Although acuminate papules at first, they soon flatten out into slightly scaly spots.

Follicular Psoriasis.—In this the primary papule is at the hair follicle and remains attached to it. The papules may be only millet-seed to hemp-seed sized, with a scaly top, or rather larger and not so convex. The eruption enlarges at the margin very little, if at all, so that the original character is preserved. The hair growth is unaffected in most cases of the usual type, although the eruption may be very thick upon the scalp; but in the acutely inflammatory cases the hair is shed profusely, even to complete denudation of the affected area.

The nails are frequently affected both in acute and chronic cases. The most common and characteristic change begins on one side at the distal end, where it is of a dirty yellow colour, thickened and separated from its attachments; this may extend all the way down, or the whole nail may be discoloured, pitted, furrowed, transversely thickened, and raised up from its bed by proliferated horny scales. The nail changes may commence at the root, distal end, or other part of the nail blade. The mucous membranes are almost exempt in psoriasis, though a few cases are on record affecting the palpebral conjunctivæ and the mouth. The so-called psoriasis linguæ is a misnomer, and although leucopathia linguæ is occasionally seen in psoriatic subjects the conjunction is accidental. Among the rarer complications are scarring, keloid, papillary development, either soft or warty, and from this even epithelioma may result. Sometimes the warty and epitheliomatous growths ensue, not directly from the psoriasis, but as the result of excessive arsenical treatment.

Etiology.—Sex has probably no influence in predisposing to the eruptions. Statistics vary in their results in different countries. In the author's hospital cases the females were as two to one, and in private, males and females were equal.

Age.—In the author's statistics of over 800 cases, 72 per cent begin before thirty years; the numbers before twelve, between twelve and twenty, and between twenty and thirty, being equal. Twenty-two per cent begin between thirty and fifty, in equal numbers in the two decades, and only 5 per cent over that age; below five years probably not 1 per cent, and it is very rare under two years, but there are a few recorded in the first year of life, the youngest being thirty-eight days, and this was said to have commenced in the first week of life (Rille). At the other extreme stands Watson's case with the first attack at eighty-five.

Heredity.—This is generally considered to play an important part in the production of psoriasis, though Bulkley and Rosenthal said that it could only be proved in 15 per cent of their cases, but it was probably in a much higher percentage. Nielssen disputes heredity

altogether, and ascribes it all to family contagion; but there are only a few isolated cases in which inoculation seemed probable, including a few cases of vaccination psoriasis and one of experimental inoculation (Destot); Ducrey's extensive series of experiments failed, and there is a general concurrence of opinion that there is very little evidence to support a theory of contagion in this disease. The practical facts remain, that if one or both parents suffer from psoriasis one of a family of several children will be nearly sure to get it; but it is seldom that several in the same family have it, a fact rather against the idea of contagion.

Excessive meat eating is said to be a cause of this disease, and vegetarianism is recommended as a preventative; but the author had as a patient a girl of thirteen with well-marked psoriasis, who was born with a dislike to meat and had scarcely touched it all her life.

Possibly after thirty excessive meat eating may be conducive to gouty symptoms, and thus to some extent be a favouring influence. Gout does not, however, play a large part in this respect, while rheumatoid arthritis, considering its comparative rarity, seems to be strongly predisposing to it. After forty, gout plays a more important part, but has to share with other factors only 16 per cent of all psoriasis cases.

A few cases have developed soon after nerve shocks from grief, anxiety, etc.; but diseases of the nervous system play a much less important rôle than Polotebnoff claims for them. Speaking in general terms, it may be said that in a large proportion of cases beginning after thirty, defects of health, chiefly in the direction of faulty assimilation or arthritic lesions, are present, while younger subjects generally appear healthy, with ruddy, clear complexions, though in a good number even of these they are not up to their own highest standard, and any depressing influence will often determine an attack. The most striking example is in psoriatic women who suckle their children; this almost always leads to a severe and extensive outbreak.

Pathology. — The pathogeny of psoriasis is unknown. One theory is that it is the outcome of a neurosis; but even then parties are not agreed, some assuming that it is a vaso-motor defect, others that it is a central or peripheral tropho-neurosis. The other theory is that it is due to an organism, doubtless schizomycetic. This is probably the correct view, but the symmetry and rapid widespread development so frequently seen postulate that the organism is in the blood when this occurs. The frequent occurrence of one or two patches remaining isolated for a long time, and then the rapid occurrence of generalisation in some cases, is suggestive of a primary invasion of the skin and subsequent absorption into the circulation, analogous to what occurs in pityriasis rosea. Cases of slow extension and of distal nail invasion may be explained by local inoculation.

The organism is probably a very common one, but only certain persons grow it, and heredity is explicable as an inherited tissue proclivity to the growth of the organism if exposed to it. Unna's view that psoriasis is the extreme end of the chain of the seborrhœic process does not meet with much acceptance.

Anatomically, the changes are those of moderate inflammation in the papillary layer of the corium with pronounced vascular dilatation; enormous increase of the horny layers with separation of the lamellæ; increased development of the prickle cell layers except over the papillæ; great down-growth of the inter-papillary processes and consequent enlargement of the papillæ.

The interpretation of these changes is a matter of controversy. While some regard the process as one of inflammation of a mild type, others say that the inflammatory changes are secondary to the rete changes, and one of the most recent observers asserts that the first change is in the horny layers, but this is improbable.

Diagnosis. — An ordinary case presents no difficulties, the symmetrical distribution predominating on the extensor surfaces, especially the elbows and knees, the discoid shape, well-defined border, and spongy crust of adherent silvery scales almost concealing the reddened base; and when the scales are picked off, the bright red, easily bleeding points which are exposed, and the dry character of the inflammation, make up a very characteristic group of symptoms.

Difficulties arise when some of the above symptoms are absent, ill-marked, or modified by various conditions.

Symmetry may be absent from some local cause, or in the early stage of the first attack, when it may be unilateral for some time. The discoid shape is often lost or obscured by coalescence. The crust of scales may be removed by washing, as on the hands, or by some application, before the case comes under observation.

In very pruritic cases, the patch may be scratched into an eczematous condition, so that it is no longer a dry inflammation, but very often only blood crusts are produced. The diseases most like psoriasis are dry patches of eczema, especially seborrhœic forms; lichen planus patches, pityriasis rosea, squamous syphilides, tinea circinata, and some scaly forms of lupus erythematosus.

The patches of eczema, even when round and better defined than usual, as in the seborrhœic form, are never thickly covered with scales, and what there are, are in a single layer, and in no way conceal the ground colour. There are no bright red points, as in psoriasis, and serous

exudation is readily produced by scratching. The ordinary weeping and crusted eczema is not likely to give rise to doubt.

Lichen Planus.—When lichen planus papules coalesce into a patch, it becomes raised, well-defined, and somewhat scaly. It is never so scaly as psoriasis; it almost always has a violaceous hue, which should suggest the idea of lichen planus, and then careful search will almost always find some of the characteristic flat, angular papules, either near the patch or in some one or more of the favourite positions of lichen planus.

In the psoriasiform seborrhœic dermatitis, the resemblance is sometimes very close in some of the patches, but the distribution is different, the axillæ, trunk, and groins are the usual seats of secretion, the scales are much less abundant, and are not in silvery crusts, but scanty and dull from the large admixture of fat, and seborrhœa capitis is almost sure to be present. The patches themselves are bright red and well defined, but are often not circular.

Pityriasis rosea.—Here, although there are well-defined scaly discs in many of the cases, they are altogether more delicate than those of psoriasis. They are very little raised above the surface, the ground colour is paler, and the scales are very fine and scanty. The lower segments of the limbs are nearly always free, and the eruption is acute in development, and disappears spontaneously in a few weeks in most cases, although in some it lasts a few months.

Squamous Syphilides.—The small, scaly syphilide of the early secondary period is the most like an early psoriasis. In the syphilide, the distribution favours the flexor rather than the extensor aspect of the limbs, although not very decidedly; the eruption is more uniformly distributed than in psoriasis. The patches are always small, seldom over half an inch, the scales are scanty and dirty-looking, the ground colour, though fairly bright red at first, soon acquires a dirty, brownish-red tint. The eruption comes out in crops, so that all stages are present together; the faded eruption leaving very marked staining. Further, it is common to find more than one kind of eruption out at the same time, and there are almost sure to be the corroborative symptoms of syphilis in the glands, mouth, eyes, hair, etc.

The circinate and orbicular syphilide is something like the circinate psoriasis. Here again the distribution is different. The syphilide is most common at the nape, forehead, and around the nose, while it is absent from the usual psoriasis positions, and seldom attacks the limbs at all. The scales are less abundant, the rings are primary, and not therefore formed from a disc clearing in the centre, and being an early eruption corroborative symptoms are sure to be present.

A tertiary, circinate, scaly syphilide more closely resembles a psoriasis lesion, but the patches are generally few in number, not symmetrical, are not in the psoriasis positions, often on the face are more heavily crusted, and when the crust is removed ulceration is often present in some of the lesions, while the centre of the patch frequently shows atrophic scarring; scars of previous lesions also are often present.

Tinea Circinata. — The small number of lesions, the want of symmetry, and chance distribution, the scantiness of the scales, the finely papular border, and the very definite circular shape of the patch or ring ought to prevent error. Scrapings well soaked in liquor potassæ would show the mycelium of the fungus.

Pityriasis rubra is only like the acutely inflammatory psoriasis, and can only be mistaken in the early stage. In advanced cases the universality of the eruption, the large, flaky, loose scales, and the intensely red surface are distinctive. In acute psoriasis there are always some areas of healthy skin, the scales are smaller and less flaky, and all the symptoms are less developed or intense.

In all the above long list of diseases presenting more or less resemblance to psoriasis, errors arise from not taking all the points into consideration, and laying too much stress on one or two points of resemblance.

Prognosis.—The prognosis is good for any one attack if efficiently treated, but it is impossible to promise a permanent cure. The interval of freedom may be short or long, a few cases being several years without a recurrence. Immunity is most likely to be secured by persevering in the treatment until every spot has been removed.

Treatment.—A judicious combination of internal and external treatment is the most rapid method of removing the eruption, although there are many cases in which either method alone could be made successful in skilled hands.

There are no known means by which recurrence can be prevented, though by keeping the patient up to the highest standard of health attacks are less likely to occur, and by treating them promptly when they do appear, the extent and severity may be minimised. None of the so-called specifics have a prophylactic action, and some are injurious at the developmental period. Complete removal of every spot gives the best chance for prolonged immunity.

Whenever any defective general health can be detected, the soundest practice is to treat that on the general principles of medicine whatever the condition of the skin may be; but inasmuch as in psoriasis under thirty there is often no defect of health discoverable, there are a certain number of drugs which experience has shown have a beneficial action on the diseased skin of many persons, although we cannot always explain their *modus operandi*.

For a long time arsenic was considered to be

the only drug which had a material effect on psoriasis, and was *facile princeps*. Of late years thyroid extract, salicin and its derivatives, and iodide of potassium in heroic doses have come in as valuable aids in the treatment of this often obstinate disease. Their indications and limitations are as follows:—

Arsenic.—While arsenic has an indubitable influence on the eruption, in many cases it must be confessed that it very often fails, and sometimes aggravates the eruption, both as regards itching and the evolution of fresh patches. In many cases the drug must be given in large doses and for a long period before any beneficial effect can be obtained. It has no prophylactic effect.

As regards the eruption, arsenic should not be given in the acutely inflammatory form, nor whenever the patches are very hyperæmic, show a great tendency to spread at the margin, or when new lesions are coming out rapidly.

Its most favourable effects are seen when the hyperæmia is moderate and the disease is stationary, or where there are only a few chronic patches.

As regards the patient, many are intolerant of it even in small doses, but while young people with good digestive organs can generally take fairly large doses for some time without conspicuous ill-effects, it should not be given to persons with an irritable gastro-intestinal mucous membrane, or with defective powers of elimination.

It may be given either in a solid or liquid form by the mouth, and rarely by subcutaneous injection.

When the solid form is chosen the Asiatic pill is the favourite mode in Germany. ℞ Arsenious acid, grs. $8\frac{1}{4}$; powdered black pepper, grs. $67\frac{1}{2}$; gum Arabic and water sufficient to make a mass which is divided into 100 pills, each containing $\frac{1}{12}$th of a grain. From one to three pills three times a day are given, in Germany, before meals, but it is much less likely to upset the stomach if taken after meals; if there is not a decided effect after 500 pills, arsenic may be considered to have failed.

In England arsenite of potash in the form of Fowler's solution is usually chosen; it has the advantage of being dilutable to any extent, and therefore less likely to upset the stomach; it contains 1 grain of arsenious acid in 110 minims, and the average dose is ♏v. three times a day, but it is best to begin with ♏iij. and gradually increase it to ♏x., according to the patient's tolerance. It should be given after meals and diluted with at least two ounces of water, and is often best combined with a bitter, such as tincture of hop, ♏xx.

If the patient can bear it, it may have to be taken for three months before striking benefit is observed; but it often has to be given up long before this, from its irritant effects on the gastro-intestinal canal, producing dyspepsia, griping, and diarrhœa, or from its producing other toxic effects, of which pricking of the conjunctiva and puffiness of the eyelids and a coated tongue are among the earliest symptoms, and should indicate a diminished dose, if not an actual withdrawal of the drug. The site of the patches is more deeply and persistently pigmented than usual in cases treated by arsenic. Patients who take arsenic for a long period become generally pigmented, while keratosic and warty thickenings develop on the palms and soles, but only in a few would such symptoms arise unless the dose was large or long continued. Epithelioma may develop on the warty thickenings many years after the arsenic has been left off, in one of the author's cases over forty years after.

Cacodylate of soda, a compound of organic arsenic, has recently been recommended as an improved means of giving arsenic in much larger doses than usual. One grain of the salt is said to be equivalent to $\frac{1}{10}$ of a grain of arsenious acid or 60 minims of Fowler's solution. Half a grain is the initial dose recommended, and it is said that as much as 3 grains a day may be taken with safety. The author, however, does not consider it a safe drug, and if given at all, $\frac{1}{16}$ of a grain three times a day is enough to begin with.

Thyroid extract as a treatment for psoriasis was introduced by Byrom Bramwell. While it has come short of what it was announced to be at first, in a moderate proportion of cases it acts most beneficially and more rapidly than anything else. It is, however, a remedy which should be given with care and under close medical supervision. It should not be given to old people or to those who have weak hearts. As regards the psoriasis, it should not be given at the onset of the disease, nor whenever the eruption is undergoing development, as it often then spreads rapidly under thyroid influence. It is most likely to be of service in the same class of cases which are indicated for arsenic, but would not be worth trying unless the eruption was extensive.

The most convenient form is the five-grain tabloid; only one a day should be given for the first week, then two a day, and if the patient is tolerant, in about ten days the dose may be raised to three tabloids a day. It is seldom necessary to go beyond this, and unless the patient is in bed and under supervision is not safe. Headache and a pulse over 120 should be the signal to reduce the dose or stop the thyroid extract altogether. If after a month there is not decided improvement the case is probably not suitable for the treatment. Iodothyrin and thyroidin are also given in smaller doses, but it is better to give the entire gland product.

Unfortunately, one can never be sure beforehand in any particular case whether it will be of service, and even in the same patient it will succeed in one attack and fail in another. This leaves only a very limited number of cases in which it will be successful, but in the best cases for it, improvement is often very rapid.

In the author's practice both arsenic and thyroid have been in great part superseded since he discovered the remarkable power of salicin and salicylates in removing the psoriasis eruption. As salicin is in most cases as efficacious as salicylate of soda, and less liable to upset digestion, it is the one to be chiefly used. It must be given in adequate doses. As a rule 15 grains three times a day in an ounce of water is the best dose to begin with, and as most people are very tolerant of it, the dose may be increased to 20 or even 30 grains, but its solubility is not more than 19 grains to the ounce, and it is better given in solution than in cachets or tabloids. In the large doses 4 minims of nux vomica tincture may be added to counteract any depression caused by the drug, but this is rarely seen. Salicin erythema eruptions are also rare, and in only a few does it disturb digestion or produce headache. It has the great advantage over arsenic and thyroid that it may be given with benefit in developing cases, and will often check the increase; and it disagrees in the above ways in so few cases that it is always worth trying, and while, of course, not always successful, it is so in a much wider range of cases than either of its rivals, with fewer drawbacks. It is more likely to produce speedy improvement in widespread hyperæmic cases than where there are a few chronic patches; it has also not much effect on psoriasis of the scalp. In suitable cases, the hyperæmia diminishes, the scaly crust becomes loose and then falls off, and gradually new scales cease to form, the patch clears up in the centre, the ring breaks up into fragments, which are best removed by local means. In the most favourable cases, this may be seen in from a month to six weeks, even when no local treatment has been simultaneously employed, but, of course, the majority take much longer. Like all other drugs, it does not act as a prophylactic against future attacks. The action of salicin is, in the author's opinion, due to its breaking up in the organism into salicylic acid, and exercising a microbicide action in the blood or lymphatic circulation. Iodide of potassium in heroic doses has been strongly recommended by Greve, Boeck, Hasland, and others, and has undoubtedly good effect in some cases. Commencing with 10 grains three times a day, in a week it may be increased to 15 grains, and when tolerance is established the dose may be rapidly increased; as much as 750 grains a day have been given. In the author's opinion better results may be obtained by other means with less injury to the patient, for iodides have often a very injurious effect upon the gastro-intestinal and other mucous membranes, and it is not a drug to be given indiscriminately, for unless the heart and kidneys are quite sound dangerous iodism might ensue. It is uncertain whether its good results are due to its diuretic action or to the liberation of nascent iodine in the circulation.

Phosphorus, carbolic acid, antimony, and turpentine occupy quite a minor place compared to the previous drugs. The two latter are better than the first two, and are chiefly suitable for very hyperæmic cases. From 5 to 10 minims of antimony wine three times a day is the usual dose, and from 10 to 15 minims of turpentine suspended in mucilage; copious diluents, a quart of barley water or more a day, being given to counteract the diminution of urinary water which otherwise ensues.

In very widely spread eruptions with great congestion, often the best treatment is large doses of quinine, gr. iij. to gr. v. three times a day, given in the acid portion of an effervescing potash mixture, gr. 22 bicarbonate of potash to 15 grains of citric acid being the usual proportion.

There are a few chronic cases which resist arsenic, thyroid, and salicin, even when combined with what is usually efficient local treatment. In these cases the intra-muscular injection of mercury is often most valuable. The author uses sozoiodolate of mercury dissolved with the aid of an equal weight of iodide of sodium; $\frac{1}{4}$ of a grain is injected into the buttock once or twice a week. Prault obtained good results in two cases by injecting yellow oxide of mercury ·05 gramme.

Mapother strongly advocates mercury salts internally for psoriasis.

Local Treatment.—This is of the highest importance, and in a large proportion of cases is sufficient by itself to remove the eruption. The indications are to remove the scales, diminish the hyperæmia, and then use a microbicide. In acute cases, when the first two indications are fulfilled, often nothing more is required. In chronic ones, when the scales have been removed, the microbicide treatment can be proceeded with at once.

The means to remove the scales are alkaline baths, ʒiv. of bicarbonate of soda to a 30-gallon bath, at a temperature of 95° F., the patient remaining in twenty minutes and rubbing off the scales after soaking. The bath may be repeated twice or thrice a week. Wet packs may be employed instead, but require the patient to lie up.

When there are only a few chronic patches, as on the knees or on the scalp, soft soap rubbed in with wetted flannel and then rinsed off is

very efficacious, but is not suitable for actively inflammatory patches. It may be repeated every day, or three times a week, according to its effect. An improvement is saponis mollis, spirit. vini rect., or eau de Cologne, partes equales; mix, and make a liniment.

In the more acutely inflammatory cases, wrapping the limb or body up in lint soaked in glycerini plumbi subacetatis ℥j., aq. distil. ℥vij., and covering it with oiled silk or hat lining, removes the scales and diminishes the hyperæmia, and will cure many patches.

Other applications suitable for acute cases, or whenever it is desired to reduce hyperæmia before resorting to special remedies, are calamine liniment, consisting of calamine gr. xl., zinci oxid. gr. xxx., lanolin ʒj., ol. olivæ and aq. calcis ad ʒiiiss., applied constantly by means of lint soaked in the liniment; or, covering the part thickly with Lassar's paste, with or without the salicylic acid, zinci oxid. ʒij., pulv. amyli ʒij., vaseline ℥ss., cover paste with butter cloth. Assuming that the hyperæmia has been removed by one of the above means, or that there is not enough to require such preliminary applications, what special application shall be selected?

All the special remedies are microbicides, and all have a more or less stimulating action. As a rule, the less hyperæmia there is, the more stimulating the remedy may be, but care must be taken not to irritate the patches too much, or the soothing treatment will have to be reverted to, and time be lost. Whatever the remedy, it must be rubbed in thoroughly to each patch, working especially round the edge. In extensive cases, an attendant may have to spend from two to three hours a day in applying the remedy, and this is the secret of success, together with adapting the remedy to the condition of each patch.

Only a few of the legion of infallible remedies need be mentioned as the survival of the fittest.

Chrysarobin stands first on account of its almost specific action on the diseased area, but it has serious drawbacks. It stains linen indelibly, and dyes the hair a purple-brown, and the nails also. It is also very liable to excite an erythema for a considerable distance beyond the point of application. This is sometimes so severe, and attended with such swelling, that it has been mistaken for erysipelas when situated on the face, where it also excites conjunctivitis. It should therefore never be used near the face, and the patient should be warned of the possibility of these disagreeables to clothes and person.

In covered places and suitable patches the result is almost magical, the disease being cleared off in a short time, leaving a white area on a red ground. Such a result is not always attainable, but a very satisfactory removal of the patch may often be obtained in a slower and less sensational manner. The stronger ointments, from gr. xx. to ʒj. to the ounce, are only suitable for indolent and pale patches. Since the author found that from one to five grains to the ounce of zinc ointment is signally beneficial in many hyperæmic cases its range of usefulness has been much extended.

When the surface is cleansed from scales, and the patch is pale, a paint may be used which is convenient to apply and does not stain; it is composed of chrysarobin gr. xx. to ʒj., chloroform[1] traumaticin ℥ix. The British Pharmacopœia traumaticin is made with bisulphide of carbon as a solvent, but its fæcal odour makes it an impracticable preparation. After painting for six days one coat over another the film may be peeled off, and a large part of the diseased area will be found to be cured. It is often advisable to wait a few days and apply an ointment before renewing the painting.

Pyrogallic acid is another valuable remedy, but it also stains, but to a less degree. It may be used in subacute cases, from 10 to 60 grains to the ounce of ointment, but it must not be used over a large area at a time, as dangerous symptoms may arise from its absorption. Dilute hydrochloric acid is said to be prophylactic and remedial for the toxic symptoms. For the face and other exposed parts those preparations which neither stain nor smell should be selected. They are resorcin, salicylic acid, thymol, which has the smell of thyme, but is cleanly, and naphthol, all gr. x. and upwards to the ounce.

Most of the mercurial ointments also are suitable for the face and for limited areas in other parts. They are the diluted nitrate ʒij. to the ounce, ammoniated mercury gr. xx. up to ʒij.; the yellow oxide ʒj. to the ℥j., is stronger; the bichloride and biniodide, gr. j. and upwards.

They should only be rubbed on, never continuously applied. The ointment base is lard, lanolin, or vaseline, or a combination of them.

Many tar preparations are most valuable, and have stood the test of time, but their smell limits their use to those who can discontinue their social or business duties. They are oil of juniper pine (oil of cade), oil of birch (ol. rusci), creasote, all from 10 ♏ to 60 ♏ to the ounce. The old tar ointment is good, but seldom used, as it is dirty as well as smelling strongly.

A 5 to 10 per cent watery solution of liquor carbonis detergens, or liquor picis carbonis, is often convenient for large areas for self-application, but is not so efficacious as inunctions. The pure solution may be used if followed by the continuous application of the glycerine of lead solution.

For the scalp, the spirit of soap liniment should be rubbed in with flannel dipped in hot water and then in the liniment, then rinsed out,

[1] To make this of proper consistence, pure guttapercha ʒj. should be digested in chloroform ʒx. for two or three weeks, with agitation.

and the ointment of perchloride or biniodide of mercury rubbed in every day.

It should be thoroughly understood that there is no routine treatment as far as drugs are concerned. Their proper selection and adaptation, not only to each patient, but to each region or even patch of disease, only comes with practical experience, and after this their application must be thorough, and the strength or medicament altered according to the effect produced. It is always wise to begin with a weak preparation and increase the strength gradually. Where the patient will give himself up to treatment with a trained nurse, and the frequent supervision of an experienced practitioner, most cases of psoriasis, however extensive, can have the eruption removed in three or four weeks. Where the patient carries out his own treatment, hammering on with one prescription, three or four months to a year is the usual period, and even then failure often results.

Many patients, however, prefer treatment at spas, and when they are situated at high altitudes the invigoration of the climate is an important adjunct. In other cases, the results are not superior to what can be obtained at home if the patient will devote as much time to the treatment. They are best for wealthy patients who cannot or will not give up their social or business engagements unless they leave home.

The baths are of two classes, thermal and sulphur. At thermal stations, such as Leuk or Aix-les-Bains, the treatment consists in prolonged immersion in weak alkaline hot waters. In sulphur baths, such as are found at Aix-la-Chapelle, Barèges, Harrogate, Strathpeffer, etc., in addition to the thermal action the sulphur acts as a microbicide. Very acute cases are not suitable for these baths, and they should always be taken under medical supervision, or they will often do more harm than good.

A few spas contain arsenic. They are: Levico, beautifully situated in the South Tyrol, near Trient, 4880 feet above the sea-level. It is the strongest arsenical spa known, and there are two sources; the strongest contains $\frac{1}{12}$ of a grain of arsenious acid to the pint and 4 grains of iron salts, chiefly persulphate. The dose is one tablespoonful. The weaker spring contains $\frac{1}{120}$ grain of arsenious acid to the pint and 8 grains of proto- and persulphate of iron. The imported waters can very well be taken, and as a matter of fact the water is brought down from a mountain behind Levico in pipes.

La Bourboule in the Puy de Dôme, France, being more accessible, is better known. The arsenic is in the form of arseniate of sodium, 28 mg. to the litre, or between one and a half to two grains to the gallon. The other constituents are lithium, sodium, and potassium chloride, sodium bicarbonate, lime, iron, and free carbonic acid. A large tumblerful is the average dose.

At Royat some of the waters are of a similar character, but weaker. The strongest is Saint Victor, which contains about one-sixth of the quantity of arsenic found at La Bourboule, but more iron.

At these spas, thermal treatment is usually combined with internal imbibition of the arsenical springs.

Psorospermosis or Psorospermiasis.—Diseases due to the presence of psorosperms or coccidia; coccidiosis. *See* Parasites (*Protozoa, Coccidiida*); Liver, Diseases of (*Calcification of*); Mammary Gland, Diseases of (*Paget's Disease of the Nipple, Morbid Histology*).

Psychalgia.—Melancholia or (literally) mental pains (Gr. ψυχή, mind, ἄλγος, pain). *See* Insanity, Nature and Symptoms (*Types of Episodic Insanity, Melancholia*).

Psychiater.—A mental physician or alienist, *psychiatry* being the treatment of mental diseases.

Psycho-. — In compound words *psycho-* (Gr. ψυχή, the mind) means relating to the mind; *e.g. psychocoma* (mental stupor), *psychokinesia* (impulsive insanity), *psychoparesis* (mental enfeeblement), *psycho-physics* (experimental psychology), and *psychotherapy* (the treatment of disease by mental suggestion).

Psychoses.—Diseases of the mind. *See* Senile Insanity (*Psychosis of Senility*); Puerperium, Pathology (*Puerperal Insanities*).

Psychroæsthesia. — The subjective sensation of cold (Gr. ψυχρός, cold).

Psychrometer.—The wet and dry bulb thermometer for calculating the dew-point (Gr. ψυχρός, cold). *See* Meterology (*Hygrometry*).

Psychrophobia. — Excessive fear of cold, especially of cold water (Gr. ψυχρός, cold).

Psychrotherapy.—The treatment of disease by means of cold.

Pterion.—The region of the skull in the neighbourhood of the lower end of the coronal suture where it comes into relation with the squamous part of the temporal bone and with the great wing of the sphenoid; a small fontenelle (*antero-lateral*) may be found here in early life.

Pterocarpia Lignum.—Red sanderswood; red sandal-wood. The heart wood of *Pterocarpus santalinus*, found in Ceylon. The chief constituent is the red crystalline principle, *santalin*. It is contained in *Tinctura Lavandulæ Composita*. It is used as a colouring agent.

Pterygium.—The adhesion of a triangu-

lar fold of the ocular conjunctiva to the cornea (Gr. πτέρυξ, a wing). *See* Conjunctiva, Diseases of (*Various Conditions, Pterygium*).

Pterygium Unguis.—A coalescence of the skin at the root of the nail with the nail. *See* Nails, Affections of the (*Pterygium*).

Pterygoid.—Wing-shaped (πτέρυξ, a wing), *e.g.* the external and internal *pterygoid plates* or processes of the sphenoid bone, between which is the *pterygoid notch*, and in the neighbourhood of which are the *pterygoid fossa*, the *pterygoid tubercle*, and the *pterygo-palatine canal*.

Ptilosis.—The loss of hair on any part of the body; *ptilosis ciliaris* is the loss of the eyelashes; madarosis.

Ptomaines.—The term ptomaines (Gr. πτῶμα, a corpse) was formerly applied to alkaloids produced by decomposition of the body tissues, but is now also applied to alkaloids found in the body during life. The term leucomaine was at one time applied to the animal alkaloids produced during life, in contradistinction to ptomaines produced by decomposition of dead animal matter. Both these terms are now more or less obsolete, and the term animal alkaloid is universally applied. The nature and source of these complex and important substances is described under "Alkaloids" in Vol. I. *See also* Cholera Nostras (*Etiology*); Digestion and Metabolism (*Intestinal Secretion, Ptomaines*); Micro-Organisms (*Products of Bacteria*); Physiology, Food and Digestion (*Fate of Digestive Secretions in Alimentary Canal*); Snake-Bites and Poisonous Fishes (*Putrefaction of Fish, Ptomaine-Poisoning*); Stomach and Duodenum, Diseases of (*Etiology, Toxic Substances*).

Ptosis.—Drooping of the upper eyelid, congenital or acquired, due to an affection of the levator palpebræ superioris. *See* Eyelids, Affections of (*Affections of Muscles, Levator Palpebræ, Paralysis*). *See also* Brain, Tumours of (*Diagnosis, Tumour of Crus Cerebri*); Conjunctiva, Diseases of (*Trachoma Symptoms*); Hysteria (*Motor Disorders, Blepharospasm*); Lacrimal Apparatus, Diseases of (*Disease of Lacrimal Gland, Results*); Myasthenia Gravis (*Symptomatology*); Ocular Muscles, Affections of (*Paralysis*); Ophthalmoplegia; Syphilis (*Tertiary, Eye Affections*); Tabes Dorsalis (*Symptomatology, Ocular Paralysis*); Thyroid Gland, Medical (*Exophthalmic Goitre, Stellwag's Sign*).

Ptyalin. *See* Physiology, Food and Digestion (*Digestion in the Mouth, Saliva, Chemical Characters*); New-Born Infant (*Physiological Peculiarities, Buccal Digestion*).

Ptyalism.—Salivation (Gr. πτύω, I spit), *e.g.* from mercurial poisoning or as a disease of pregnancy. *See* Pregnancy, Affections and Complications (*Digestive*); Salivary Glands, Disorders of (*Ptyalism*).

Ptychotis Oil.—This oil, known also as Ajowan oil (*Oleum Ajowan*) is obtained from the fruit of *Carum copticum*, and is used for the same purposes as thymol (carminative, antiseptic, etc.); it is official in the Indian and Colonial Addendum to the B.P. *Dose*—½ to 3 m.

Puberty.

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See also Adolescent Insanity (*Insanity of Puberty*); Cough (*Barking Cough of Puberty*); Epistaxis (*Etiology, Puberty*); Larynx, Neuroses of (*Nervous Laryngeal Cough*); Menstruation and its Disorders (*Puberty*); Mental Deficiency (*Cretinoid Cases, Absence of Puberty*).

Puberty is the time at which the reproductive organs enter the initial stage of potential or physiological activity. It is the beginning of the development from childhood into maturity. The time occupied in this development is long, in most people 12 or 13 years. It is known as adolescence, and it may be said to begin normally at from 12 to 14 or 15 years of age with the appearance of the first signs of puberty, and to end at the age of 25. As W. S. Christopher puts it, adolescence is the "epoch for the latentisation of force for reproductive purposes, and this latentisation requires a high nutrition and a relatively low activity."

In childhood the whole energy of life is directed towards growth, the building up of the bodily frame, bone, muscle, nervous tissue, etc. Children are more ready to suffer from malnutrition than adults, as they have not only to live but to grow, and the daily demands made on their nutritional powers are necessarily much greater than they are when growth has ceased. Long before young people have done growing the organs of reproductive life pass from quiescence to a certain measure of potential activity. From the date of this change, which we call puberty, the child has not only to live and to grow, but to develop his highest mental powers as well as his most complex animal functions. As perfect function cannot precede complete growth, it is clear that puberty does not mark the time at which the functional activity of the reproductive organs would be anything but harmful. Nature's care for the species rather than for the individual is shown by the age at which reproduction is possible. From the point of view of the individual the premature exercise of the generative functions is opposed both to perfect growth and to high development. Adolescence should be entirely occupied with growth

and with the development of mind and body to the highest perfection possible to the individual.

Signs of Puberty.—In boys the "breaking" of the voice is one of the earliest signs of puberty. A boy's singing voice usually resembles a woman's soprano, but the note is less flute-like, more vibrant, and of clarionet quality. At puberty the larynx elongates, the vocal cords are at first relaxed, the voice is for a time hoarse. Then readjustment takes place, and the man's voice appears, usually an octave lower than the boy's. The speaking voice goes through similar changes; it becomes hoarse, and finally much deeper than in childhood. Concurrently the external genitalia grow and become more vascular. A considerable development of hair takes place on the face and pubes, and in a less degree on the arms and chest. Connected with this is a great development of the sebaceous glands of the skin, leading in many cases to an eruption of acne on the face and shoulders.

In girls menstruation is usually the first and most important sign of puberty. It occurs normally at varying ages from 11 to 16. As a rule it appears about 13. Within what may be called normal limits its early appearance is a sign of vigour, its postponement a sign of retarded and imperfect development, or of poor nutrition. Women of the best physical type are generally in advance of their age in youth and behind it in middle life. Menstruation in them begins early, it is painless and moderately profuse, and it lasts till 50. It is quite exceptional for menstruation to be painful for the first year or more of its occurrence. Dysmenorrhœa usually begins eighteen months or more after the first appearance of the catamenial loss. For a time, in most young girls, menstruation is the only sign of any considerable physiological change. They are still children in thought, feeling, and tastes. It is difficult to determine how much of the gradual change from childhood to adolescence is really due to the changed condition of the organs of the reproductive system, or how much of it ought rather to be attributed to the development of the nervous system generally and to the increasing influences of education and social and family life. In the opinion of the present writer a good deal of exaggeration is present in the writings of physiologists on this point. Girls of 14 and 15 are not, as a rule, troubled with anything that can be called sexual feelings. They are practically as free from them as they are at 7 or 8. Moreover, in the small minority of young women under observation who have never menstruated, and who presumably have either no ovaries or very undeveloped ovaries, the writer has not been able to recognise anything exceptional in their demeanour, in their desire for sympathy, affection, or admiration, or in their appreciation of pretty garments. If the entire mental organisation of women were based upon sexual development, as some writers would have us believe, women who are without the most important part of the reproductive system ought to be much more unlike other women than they actually are. Similarly, in the curious type of women who may fairly be called "masculine"—the women who are genuinely, and from no affectation of their own, really like men in their habits of mind, their virtues and their defects—the writer has not been able to trace anything abnormal on the sexual side of their bodily development. On this side they are real women; on every other side they closely resemble men. It has to be admitted that the resemblance is a genuine one, and not the result of any "apeing" of men. Many women of this curious type have done their utmost to be ultra-feminine in dress, etc. We all remember Sydney Smith's inquiry, "Who is the gentleman in white muslin on the sofa?" in reference to a well-known woman who strove through a long life to be like the sex she belonged to physiologically, but not mentally. If the feminine qualities and characteristics are based solely upon the sexual organisation it is difficult to understand cases where mind, general formation of body, and temperament seem to belong to one sex, and the special distinguishing organs of sex to the other. They seem to suggest that the connection between the sexual system and the bodily and mental characteristics usually accompanying it may be less direct and immediate than is commonly supposed. In the two above-mentioned groups of what may be called "freaks" women are feminine without the fundamental feminine organs, or they are essentially masculine in spite of possessing them. Some correlation, which usually connects the sexual type of mind and body with the sexual organs, seems to be absent.

Precocious and Retarded Puberty.—Puberty is occasionally precocious in both sexes. In boys very marked precocity has often been part of general imperfection, both mental and bodily, as in Dr. Campbell's and Dr. Wood's cases. In girls it is not rare for menstruation to appear long before the usual date of puberty. In many of these cases the phenomena of menstruation stand alone and are not accompanied with other signs of precocious maturity. Children of from 1 to 7 years of age have had regularly recurring menstrual loss without any obvious abnormality of development. In another class of case, however, there has been seen very remarkable growth and size with development of the breasts and labia, and then precocious menstruation. Here there has been a physiological anomaly of development of which the precocious menstruation was but a part and a sign. Dr. Morse, of Harvard, has collected and published 30 cases of these two groups of precocious children. Among these there was one

child of nine months old, another of seven months, who menstruated regularly. Both were abnormally large and intelligent even from birth. A post-mortem on one case showed ovaries which had ovulated regularly. One child, whose case is given by Dr. Morse, became pregnant in her eighth year, another at ten, and a third at eight and a quarter. In all these cases there was nothing, strictly speaking, abnormal except the precocity. They were anomalies of development. Where precocious menstruation is present without any general early maturity, the cause of the condition is even more obscure.

Cases are not uncommon in which, from change of climate, nervous shock, a severe illness at or about the time when puberty should have occurred, or from other causes, the normal rate of development has been checked, and menstruation is considerably delayed. As a rule, in these cases, if there is good evidence of a sound general development, *e.g.* in breasts, pubes, etc., there is reason to expect menstruation to appear. If at 20 or more than 20 the breasts, etc., are infantile, it is probable that the ovaries are absent or imperfect. Where the ovaries are present, though functionless, menstruation may occur for the first time even at a very late age. Dr. Wolfe reports a case in which the patient menstruated for the first time at 43. She had been treated for some months when 19 years of age for amenorrhœa. Some years afterwards she married a widower with a family, never menstruated till eleven years later, when she was nearly 43. A severe fright from an idiot girl was followed at once by menstruation. This recurred irregularly for eighteen months, when she became pregnant. In this case the fault must have been nervous. From some obscure cause the ovarian nerves were inhibited, possibly in a way analogous to the inhibition of common sensation, or of the special senses, in hysterical or in hypnotic conditions.

Disturbances of Health at Puberty.—In the great majority of cases the commencement of the change from childhood to adolescence involves no disturbance of health. Where the nervous system is sound, the nutrition good, and the habits of life wholesome, health may be expected at this as at other ages. This particular step towards maturity ought not to involve any more risk than the many previous steps from infancy upwards have done. Experience confirms this view. Every mother knows that a sound child of from 13 to 16 is a very much more robust creature than he or she was ten years earlier. He is much less often ailing, his life is more secure. She takes it for granted that the changes which puberty indicates will be accomplished without nervous or other disturbances, and in the case of healthy children her hope is almost always justified by the result. The case is, however, quite otherwise when the child inherits a bad nervous system, or when its nutritional powers are unequal to any extra stress being put upon them. Related to the fundamental changes accomplished at puberty, there is the correlative though not usually quite contemporary change of very rapid growth, and of an equally rapid development of the higher mental powers. Nervous stability is taxed in several ways at once. These conditions, however, belong more properly to adolescence than to the time of puberty itself.

In both boys and girls the time of greatest rapidity of growth is later than puberty, and it is probable that very rapid growth has more to do with the nervous instability of adolescence than anything else. Insanity is indeed rare at puberty, and markedly less rare six or seven years later. When present it is always the result of a strongly marked inherited tendency. Dr. Clouston had only 2 cases of insanity at puberty in the Edinburgh Asylum out of a total of 1800 cases, and only 22 at the ages of 16 and 17. He believes insanity at puberty to be always hereditary, acute, remittent, not dangerous to life, and of good prognosis.

Of the less serious neuroses, epilepsy, chorea, and migraine are the most important, and they are also among the disturbances which most frequently seem to begin at or near puberty. Especially is this true of migraine and epilepsy. A large proportion of well-marked cases of migraine date from the age at which puberty usually appears. In women it may possibly be connected with the heightened arterial tension before menstruation, and with disturbance of the vaso-motor system as a direct result of the loss of blood. It is very unusual to hear of a true migraine beginning in adult life. In previously healthy girls the occurrence of menstruation, when the loss is normal in amount and regular in its return, seems to produce no evil effects. If, however, the loss is excessive, or very scanty, or quite irregular, the general health is usually affected disadvantageously, coincidentally, perhaps, rather than consequently. The appetite often fails, and then, at a time of very rapid growth, the nutritional power is diminished and anæmia results. The power of assimilating a liberal allowance of nutritious food is never more valuable than in a girl of 15 or 16, when, along with a marvellous rate of growth, the organs of reproduction are being gradually matured and perfected, and their nervous mechanism elaborated and, as it were, trained into harmonious working. For all this a high degree af nutrition is wanted. The change started in girls at puberty, without suffering and without any disturbance of health, is frequently checked a few years later by conditions which unfavourably affect the nervous system or the nutrition. A familiar

instance of this is the bad effect on a girl's health which so frequently results from sending her abroad at 16 or 17 years of age. A visit to Germany especially very often upsets a girl's health to a very serious extent. The food is distasteful, the houses are hot and close, there is very little outdoor life, very few games, and she is home-sick and solitary. The nerves connected with the system then in process of development are often the first to break down as a result of poor nutrition and a generally depressed vitality. Mothers are apt to be unduly anxious about the first appearance of menstruation, and not nearly anxious enough to maintain vigour during the important years which immediately succeed it.

Hygiene of Puberty.—No special rules are required at this age for either boys or girls. They both need a healthy, active life, with much open-air exercise, plenty of good food, and plenty of occupation. The discipline and brightness of a good school are, as a rule, much better for girls as well as boys than the indulgence and absence of systematic playground life which are involved in education at home. All good girls' schools of the present day make almost as much use of playground games as boys' schools do, and with similarly good results. Young people of that age are best with very little leisure, with their whole time so occupied with a disciplined routine of work and play that there is no opportunity for lounging about and sentimentality, and with a large amount of social and amusing exercise. Girls who enjoy out-door games usually eat well, and if they do Nature will take care of the due development of their reproductive organs. The danger of too much exercise, especially when it is out of doors, is far smaller than the danger of too little, and the regulation of exercise may safely be left to the discretion of the schoolmistress if she is fit for her work.

Parents are often puzzled as to how far it is well to explain the elementary facts of the physiology of reproduction to girls and boys at about the age of puberty. Much depends on the relations between the parent and child; some parents are much more intimate with their children than others. A certain degree of reserve on such subjects ought certainly to be maintained; but it is probably always best for fathers to speak to sons, and mothers to daughters, in a way which shall enable the child to understand the elementary facts of life, and to look upon them with reverence. The study of botany is perhaps the best way of introducing to children the idea of sexual differences, and from botany the step can be taken to the natural history of animals, and so to human physiology. It is certainly desirable for boys to be warned as to the great danger of loss of self-control, and of any premature and irregular exercise of the reproductive function. Girls, as a rule, need such warning far less than boys, and it must depend on the discretion and insight of their mothers to give it or to withhold it.

Pubic.—Relating to the pubes (region corresponding to the pubic bones); *e.g.* the *pubic arch*, the *pubic bone* (*os pubis*), the *pubic spine*, the pubic segment of the pelvic floor, etc.

Pubiotomy or Pubeotomy or Pubotomy. *See* Hebotomy; Labour, Obstetric Operations (*Symphysiotomy*).—John Aitken, of Edinburgh, writing in 1786 (*Principles of Midwifery*, 3rd edition, p. 83), proposed an operation closely resembling the modern hebotomy or pubiotomy. "May not," he wrote, "Cæsarean section and embryotomy be superseded by a pelvitomia nova? viz. two incisions, one on each side, reaching to the ossa pubis, as near the crural vessels as safely may be, so that one may be distant from the other about four inches; and two corresponding to and touching the joinings of the rami pubis and ischiorum. The bones are then divided by the flexible saw, without wounding the peritoneum, bladder, urethra, or vagina. Thus the segment of the pelvis becomes movable, and yields to the pressure of the child so as to allow delivery. If due attention be paid to the wound, may not the healing take place in such sort, that in future sufficient capacity of the pelvis may be preserved? I am just now employed in trying the effect of this operation on brutes."

Public Health. *See* Abattoirs; Air, Examination of; Air-Borne Diseases, Anthrax; Canal Boats; Cattle; Disinfection; Epidemiology; Food; Hospitals; Infection; Meteorology; Midwives; Milk; Nursing; Pregnancy (*Management*); Quarantine Rabies; Sewage and Drainage; Trades, Dangerous; Ventilation and Warming; Vital Statistics; Water.

Pubo-.—In compound words *pubo-* (Lat. *pubes*, the hair which appears on the body at puberty) means relating to the pubic region or bone; *e.g. pubo-femoral*, *pubo-vesical*, etc.

Puccinia.—Mildew (*Puccinia graminis*), is a fungus which may grow on flour and meal.

Puddings. *See* Invalid Feeding (*Diet during Convalescence, Puddings*).

Pudenda, Ulcerating Granuloma of the. *See also* Venereal Disease (*Ulcerating Granuloma of the Pudenda*).—The distribution of this disease is not yet fully known. It occurs in British Guiana, where it attacks the pudenda of the coloured population and half-breeds. Women are rather more frequently attacked than men. Dr. Daniels states that he has seen a similar disease in Fiji

amongst the Melanesians from the New Hebrides and Solomon Islands. It was there a chronic disease, unaffected by antisyphilitic treatment, and was locally contagious, or at least affecting opposed surfaces. The present writer met with probably a dozen cases in Darfour and in the Bahr-el-Ghazal district of Central Africa. Dr. J. H. Conyers, who has had experience on the Gold Coast of West Africa, never saw the disease there. Dr. Kennard divides the affection into three classes—specific, tubercular, and malignant—but it is probable that these are only phases of the same disease. The disease attacks individuals from adolescence to old age. It is rarely, if ever, cured, and specific treatment has no influence upon its progress, although it may be useful in cases which are complicated with syphilis. Fifty years ago the disease seems to have been exceedingly rare.

There is no evidence that this affection is hereditary.

The disease usually commences as a simple pimple situated somewhere in the groin or behind the scrotum, or somewhere on the genitals. There is great itching, and buboes form. The patients scratch themselves, and the tissues break down and discharge a foul-smelling purulent fluid. An ulcer rapidly forms, having abrupt edges which are more or less indurated. There is little or no congestion of the surrounding skin. The affection may either begin on the skin or the mucous membrane; it does not affect the glands, bones, or subjacent tissue, but spreads in the dermal and sub-dermal tissue. It has a marked preference for the neighbourhood of the hair follicles and for the sebaceous and sudoriferous glands, at any rate in its advanced condition. The disease does not markedly affect the general health, and is not associated with any particular diathesis.

Microscopically the disease appears to be allied to lupus erythematosus, but there is a want of symmetry, and it is more superficial in character than that disease. It spreads very slowly—for instance, in a case which was seen in 1890 it was situated behind the scrotum, in 1892 it had spread round the anus, and in 1896 it had extended backwards to the coccyx.

No characteristic micro-organisms have been found in connection with this affection, simply micrococci staining deeply by Gram's and Löffler's methods. The nodules which exist on the margins of the ulcers are composed of round cells with large but usually badly stained nuclei contained in a delicate reticulum of fibrous tissue, which is covered by epithelium. The nodules merge gradually into a subjacent, dense, fibrous stroma, in which small masses of similar rounded cells are embedded. The growths are very vascular and the capillaries much dilated, but there are no hæmorrhages (J. Galloway).

Treatment. — This is very unsatisfactory. Tonics if necessary, but, as before mentioned, specific treatment is without effect. The local treatment consists of great cleanliness and the application of caustics. Salicylic acid, 30 or 40 grs. to creasote ointment 1 oz., may be used. This causes severe pain for one or two hours, but it does not cure the patient, as, if the treatment is stopped, the ulceration proceeds. Scraping the diseased part with a Volkmann's spoon is sometimes useful, but the only chance of cure is the free excision of the initial pimple. Usually the disease has progressed too far for this when the patient comes under observation.

Pudendum. — The external genital organs of the human female (Lat. *pudere*, to make ashamed); it is generally used in its plural form, *pudenda* (*q.v.*).

Pudic.—Relating to the pudenda, *e.g.* the *pudic artery* and the *pudic nerve*.

Pueblo. *See* Therapeutics, Health Resorts (*American*).

Puer.—A mixture employed in leather-making; it consists of water, pigeon's manure, and dog's dung, and in it the pelts are immersed; it may constitute a nuisance and require to be remedied.

Puericulture.—The branch of preventive medicine which deals with the procreation of healthy and well-formed children (Lat. *puer*, a boy, *cultura*, cultivating), especially by caring for the health of the mother during pregnancy; antenatal therapeutics.

Puerpera.—A recently delivered woman, or, less often, a woman in labour. *See* Puerperium.

Puerperal.—Relating to or occurring in the puerperium, *e.g.* puerperal sepsis, puerperal insanity, etc. *See* Puerperium, Pathology of the.

Puerperium.—This subject is described in the following sections:—

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See also Brain, Affections of Blood-Vessels (*Cerebral Embolism and Thrombosis, Causes*); Glycosuria (*Puerperal Glycosuria or Lactosuria*); Lung, Tuberculosis of (*Complications, Puerperium*); Lungs, Vascular Disorders (*Pulmonary Embolism, Causes*); Miliaria (*Miliaria Crystallina in Puerperal Fever*); Nephritis (*Chronic*); Nerves, Multiple

Peripheral Neuritis (*Puerperal Neuritis*); Pulse (*Arterial Pressure in Puerperal Eclampsia*).

Physiology

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The puerperium is the period of recovery from childbirth, and is also known as the "lying-in." It may be said to begin with the expulsion of the placenta, and to comprise clinically the first month and anatomically the first six weeks after delivery. It possesses a twofold interest: from the physiological and anatomical standpoint, remarkable changes in the maternal system supervene on the completion of labour; from the clinical standpoint, the management of the puerperium requires the most careful attention, in order to shield the patient from present danger and to avert harmful results in the future. This natural division of the subject will be followed here, on the lines of the index.

I. Changes in the Maternal System

The rapid transition from the condition of carrying and nourishing a child *in utero*, with the modifications of nutrition, circulation, and excretion entailed thereby, to the condition in which mother and child have an independent existence, has a marked influence on the whole maternal system; and this influence is accentuated by the changes incidental to the progress of involution and the establishment of lactation.

A. *Physiology of the Puerperium.*—The beginning of the puerperium is characterised by the effects of the effort of labour. Fatigue, which may amount to prostration, is combined with the feeling of rest and relief following exertion and pain; the skin, pulse, and temperature testify to the recent muscular efforts, and the patient is usually content to lie in a condition of bodily and mental inactivity. The immediate and later systemic changes now demand our consideration.

1. *Temperature and Skin.*—Immediately after delivery there is a rise of temperature, which is usually slight, but which varies with the characters of the labour, and especially with its duration. From some observations on 600 cases at the General Lying-in Hospital, made for the purpose of ascertaining what relationship existed, I found (1) that the duration of the first stage of labour has only a slight influence on the subsequent temperature, except in those cases where the membranes rupture very early; when this occurs, and the first stage lasts one or several days, the temperature after delivery is often high, even when the second stage is rapid.

(2) The influence of the duration of the second stage is more marked; the longer the second stage, the higher is the temperature after labour. The time of day at which delivery takes place has a slight influence on the temperature, but less than is attributed to it by Schroeder. To estimate this factor under conditions as similar as possible, I analysed 300 normal cases of multiparæ in which the second stage of labour lasted less than an hour. Dividing the twenty-four hours into periods of four hours each, 50 cases were taken of delivery within each period, and the average temperature in each group of 50 cases was obtained, coming out as follows:—

Hour of delivery:		Temp.
12 night to 4 A.M.:		99°
4 A.M. to 8 A.M.:	,,	98·6°
8 A.M. to 12 noon:	,,	98·6°
12 noon to 4 P.M.:	,,	98·7°
4 P.M. to 8 P.M.:	,,	98·9°
8 P.M. to 12 night:	,,	98·6°

After the administration of chloroform during labour the subsequent temperature is relatively low, even when the second stage has lasted long, and has been terminated by forceps.

The temperature during the remainder of the lying-in period presents no difference, in normal cases, from that of an ordinary healthy person.

It was formerly taught that during the first twelve hours the temperature rises, that it falls during the next twelve hours, and that, after remaining stationary thereafter for twenty-four hours, there is a second rise, corresponding to the development of lactation. This rise was said to last two days, and to then give way to the ordinary temperature, which was somewhat higher than that of perfectly healthy persons,

reaching a maximum of 100·5° F. (38° C.). So also E. S. Tait found that the highest temperature occurred on the third day, and he attributed the rise to the onset of lactation. The teaching of modern text-books is that, while the temperature of the lying-in woman is easily disturbed, it is not normally different from that of a healthy person. Galabin was among the earliest to express this view. He says: "It does not appear that any notable rise of temperature should be regarded as necessary, or absolutely normal; and the elevation about the third day is probably due, much more often than was formerly supposed, to a slight septic or traumatic disturbance." Recent American, French, and German opinions are in accord with those of English writers on this point. Varnier states categorically that "the so-called milk-fever is nothing else than an attenuated form of puerperal infection." It is not necessary to multiply quotations, and I will content myself with supporting the modern view on the careful observations of Probyn-Williams and Cutler. Observations made on 100 consecutive uncomplicated cases led them to the conclusion that the average temperature during the lying-in varies between 98° and 99° F.

Rupture of the perineum was in the former days a frequent cause of rise of temperature. Tait found that, in 23 cases of more or less severe tear, the average highest temperature was 103·8°; in 7 cases of slight tear, 102·6°; and in 11 cases of labial tear, 103·7°. With a better asepsis these temperatures disappear. Probyn-Williams and Cutler found that, in 15 consecutive cases of rupture of the perineum, requiring one or more sutures, the average of their temperatures did not differ to any appreciable extent from the normal.

W. S. A. Griffith considers that normal cases are those in which the temperature does not exceed 99°, and finds that on this basis about 60 per cent of cases may be regarded as normal.

There are two reasons why the belief in "milk-fever" should have prevailed so long. In the first place, before antisepsis was introduced into midwifery, a mild degree of sapræmia or of septic infection often occurred; and the usual time for the manifestation of these conditions is from the second to the third day, *i.e.* about the time that the breasts are taking on a fuller activity. In the second place, as Griffith points out, there is a direct connection between septic conditions of the uterus and a form of mastitis; and pyrexia, associated with a blush on the breasts, may be, and often is, due to some putrid clot or portions of placenta.

The skin of healthy lying-in women is more moist than in the non-puerperal condition, and indeed this is a feature that persists during lactation. It is not, therefore, the result of the muscular efforts incidental to delivery. It is probably correlated with the activity of the breasts, to which the glands in the skin have an ancestral relationship. No doubt an additional cause for increased activity in the skin is to be found in the fact that the skin shares with the kidneys the duties of increased elimination of waste tissue-products resulting from involution.

The pigmentation of the skin brought about during pregnancy usually fades fairly rapidly, and may have disappeared almost completely in a few weeks. The striæ lose their reddish tinge, and eventually become white and opaque.

The development of mammary functions by the skin of lying-in women was first described by Champneys, who found that in 27 out of 377 women confined at the General Lying-in Hospital, lumps appeared in the axillæ, from which a milky secretion could be expressed. In half the cases they were bilateral. These lumps are situated in the skin, and, unlike lymphatic glands, can be raised and isolated from deeper structures. They are soft, flattened, without obvious nipple, pore, or duct. They are limited to the hair-covered surface, and range in size "from the smallest perceptible to that of an egg." They are commonly first noticed on the third or fourth day after delivery, and their course coincides generally with that of the breasts, enlarging and becoming tense and sometimes tender about the third day. They sometimes enlarge, and occasionally become painful at the beginning of a subsequent pregnancy, or during menstruation. The secretion does not flow spontaneously; but when squeezed out it appears successively as granular *débris*, like the secretion of sebaceous follicles; then colostrum, and then milk. At first these lumps were thought to be associated with sebaceous glands, but a later investigation by Champneys and Bowlby has shown them to be modified sweat-glands, the sebaceous glands being unaltered.

2. *Circulatory and Respiratory Systems.—The Heart.*—During the later months of pregnancy the heart usually becomes displaced slightly to the left; but with the emptying of the uterus and the diminution of intra-abdominal pressure, it is able to resume its normal position. The base of the thorax at the same time tends to return to its normal width; it does not, however, do so entirely, except under the influence of artificial compression. A second important change is the disappearance of the hypertrophy that is found during pregnancy; this involution of the cardiac muscle is commonly associated with a soft blowing murmur replacing the first sound as heard at the apex. This was first described by Angel Money, and has been confirmed by later observers. Dakin found it to be present in 57 out of 100 cases specially examined at the General Lying-in Hospital.

The Pulse.—Retardation of the pulse during the lying-in was incidentally noted by R. Whytt in 1765. Montgomery recorded, in 1857, three cases in which the pulse was 40 to 44 on the

day after delivery, rising about 10 beats a day till it returned to normal. Two of the cases were multiparæ, and one a primipara. He mentioned that he had seen many other cases of this phenomenon, of which he was unable to offer any explanation. M'Clintock, in 1861, stated that, in a large proportion of cases of normal convalescence, the pulse scarcely deviated at all from the standard of health: with the reaction succeeding to the shock of parturition, and with the coming of the milk, there might be a rise of a few beats; but even this temporary acceleration was more frequently absent than present. He continually met with cases in which the pulse did not exceed 72 all through childbed; and many other cases he had met with where it was as low as 60, 54, 48, and even 44. But this morbid slowness he regarded as constitutional, and not depending on any condition peculiar to childbed; and he added that these patients recovered well.

A more detailed study of the question was made by Blot in 1864. The following is a *résumé* of some of his conclusions:—Slowing of the pulse is generally observed in *healthy* lying-in women. The patients on whom he made his observations had, apart from the puerperal state, an ordinary physiological pulse-rate. Most frequently the pulse-rate oscillates between 44 and 60; three times he saw it fall to 35. The slowing is not influenced by diet, by uterine contractions, or by the duration of the preceding labour; and it persists during the time often improperly described as that of "milk-fever." It is more often found in multiparæ. It generally begins in the first twenty-four hours after labour; becomes more pronounced, then remains stationary, and finally disappears gradually. The duration of the slowing is usually from 10 to 12 days. It is a very favourable prognostic sign. It is not to be explained by nervous exhaustion, for sphygmographic observations undertaken in conjunction with Marey showed that it is related to the increase in arterial tension which follows labour. Louge, whose conclusions agree in the main with those of Blot, describes three types of the puerperal pulse: in the first type it oscillates between 72 and 80, and is never slower; in the second the general rate is 72 to 80, but it may drop at times to 60 or 58, returning to the normal; in the third type the pulse becomes gradually slower, till it oscillates between 46 and 50, the maximum slowing being on the seventh day. Olshausen found that in 348 cases the pulse-rate was as follows:—

Over 60 in	128	cases, or	36·8 per cent
51-60 in	164	,,	47·1 ,,
41-50 in	53	,,	15·3 ,,
40 or under in	3	,,	0·8 ,,

Hence a pulse below 60 was found in 63 per cent of cases. Falaschi described it as occurring in 25 per cent of cases; Louge found it in 33 per cent of multiparæ, and in 25 per cent of primiparæ. Blot, on the other hand, considered it to be the general rule. The above conclusions have become crystallised in the teaching of most of the text-books of midwifery. Thus Spiegelberg says: "The pulse-rate, which had increased during labour, diminishes immediately after it, then rises again, and on the second, third, or fourth day becomes markedly slowed. The rate then varies between 44 and 70; indeed, a frequency of less than 40, even of 30, has been noticed; the usual figures are 44, 48, and 56."

Within the last few years the older teaching has been challenged, notably by Probyn-Williams and Cutler, and by Heil. From an investigation on 100 cases at the General Lying-in Hospital, Probyn-Williams and Cutler found that the average pulse-rate during the puerperium was never lower than 72, and was often nearer 80 than 70; not one case presented features corresponding to the above description by Spiegelberg. On examining the records of 20 cases at Queen Charlotte's Hospital, their conclusions were confirmed. Their 100 cases presented the somewhat curious feature that the pulse-rate was higher in the morning than in the evening. Heil's investigations also comprised 100 cases; he found that in only 12 per cent of cases was the pulse-rate less by at least 10 beats during the lying-in than during pregnancy. He concludes that it cannot be admitted that there is during the puerperium a physiological retardation of the pulse to anything like the extent generally taught; for though the pulse may be slow, the same thing may occur during pregnancy or in ordinary health; it is in no way characteristic of the lying-in period.

In the face of these conflicting views, Varnier set himself to investigate the matter afresh; and his observations led him to the conclusion that in 72 per cent of cases there was an undeniable slowing of the pulse during the lying-in. We must necessarily conclude from these diverging results that the pulse-rate during the puerperium presents great variations, depending no doubt in some degree on varying conditions of observation. Investigations on a more extended scale are required. For the present, the view that most nearly expresses the results of the majority may be considered to be this: that *during the lying-in period the pulse-rate is frequently lower than in the non-puerperal state, and almost always lower than during pregnancy; the most common pulse-rate is* 60 *to* 70, *and the instances in which it is lower than* 50 *must be regarded as quite exceptional.*

Until slowing of the pulse has been unequivocally demonstrated to be the general rule, it is perhaps futile to discuss at length the cause of such slowing. It will suffice to enumerate some of the views propounded.

Thus it has been attributed to the mental and physical rest of the puerperal woman; to impoverishment of the blood from abundant losses; to increased arterial tension (Blot, Marey, Hémey); to the horizontal position (Fritsch); to altered innervation (Löhlein); and lastly to fat in the blood derived from the involution of the uterine muscle (Olshausen).

With regard to arterial tension during the puerperium, there is again a diversity of opinion. Fritsch stated that it is diminished, and Meyburg confirmed this view; on the other hand, Probyn-Williams and Cutler support the teaching of Blot and Marey that it is usually raised. The differences of opinion may be due in some measure to want of discrimination between the immediate effect of delivery on the tension and the condition when that effect has passed off. According to Probyn-Williams and Cutler, the tension varies considerably during the first twenty-four hours after delivery, being sometimes increased and sometimes diminished; but at the end of twenty-four hours it has always increased so as to equal, or more often surpass, the tension during labour. The duration of the increase varies, but is usually longer in multiparæ. A rise of temperature, even though slight, rapidly reduces the tension.

The œdema and the *varicose condition of the veins* in the vulva and legs, which are so often present in the later months of pregnancy, usually rapidly subside after delivery.

The respiratory rhythm is said by some writers to become slowed during the puerperium. In Probyn-Williams and Cutler's cases this was not the case; the average rate was 20 to 22, and followed the pulse-rate in being higher in the morning, rather than the temperature, which was higher in the evening. The capacity of the lungs is not usually altered; for although the diaphragm has a greater range of movement after the emptying of the uterus, this is neutralised by the narrowing of the base of the thorax, which had been expanded during pregnancy.

3. *The Digestive System.*—For the first few days after labour there is a loss of appetite, which no doubt has a physiological relation to the diminished demand on metabolism, when the mother no longer has to nourish the fœtus *in utero*, and before the new claim of lactation is established. On the other hand, thirst is increased owing to the rapid elimination of fluids by the skin and kidneys. The action of the bowels is nearly always sluggish; this is attributed to the patient's quiescent condition, to the small quantity of food taken, to the increased activity of the other emunctories, and to the relaxation of the abdominal walls. Another factor which, it seems to me, cannot be ignored, is the diminished intra-abdominal pressure, which results in a temporary vaso-motor paresis; as a consequence of this the innervation of the intestines remains in abeyance, and the "abdominal pool" becomes filled by dilatation of the vessels in the splanchnic area.

4. *The Excretory System.*—For about the first week after labour the quantity of urine secreted is increased. This increase relates mainly to water, for in respect of solids the total quantity eliminated shows no increase. Thus the absolute quantity of urea and of chloride of sodium is unaltered, whilst there is an actual decrease in the quantity of phosphates and sulphates. The specific gravity ranges about 1010 to 1015. Peptone, derived from the uterine involution, is commonly present for several days. In view of the researches of Vicarelli and Knapp on acetonuria in pregnant women, which led these investigators to the conclusion that the presence of acetone in the urine was pathognomonic of the death of the fœtus, it is interesting to note that Mercier and Menu found acetone in the urine of three out of six normal puerperal women. There has been a considerable discussion as to the presence and significance of sugar in the urine since this phenomenon was first demonstrated by Blot in 1856. Blot held that sugar existed normally in the urine of all lying-in and suckling women, and in a certain number of pregnant women, and considered that the sugar gave an indication as to the quality of the milk. At first these views were not universally accepted. Wiederhold (1857) ascribed the reaction to mucus, and Leconte (1857) to uric acid. Riedel (1858), from the examination of eleven puerperal women, concluded that sugar was not normally present in the urine. On the other hand, Kirsten (1857) confirmed Blot's results, except that he affirmed that the amount of sugar was inversely proportional to the quantity of milk; and Brücke (1858) concluded that sugar was normally present in the urine not only of the nursing woman, but also of the healthy man; whilst Iwanoff (1861) took an intermediate position, saying that sugar was frequently but not constantly found. Lecocq (1863) held much the same view as Brücke, except that he thought that, whilst sugar was normally present in the urine, it was found more abundantly in the urine of nursing women. Chailley (1869) found sugar in 3 cases out of 13. De Sinéty (1873) showed that glycosuria could be produced at will by sudden suppression of lactation, and that it disappeared when the secretion and excretion of milk were balanced; and in a later communication (1876) he showed that the sugar might have other than a mammary origin, for the veins of the gland contained less sugar than the arteries. Hempel (1874-75), from an analysis of 12 cases, concluded that sugar was always present at some stage of lactation; but Gubler (1876), Johannowsky (1878), and Verneuil (1882) returned to the view of Kirsten, saying that sugar was only

found when lactation was interrupted. Kaltenbach (1877), Spiegelberg (1877), and Hofmeister (1878) all found that sugar was a constant constituent of the urine of lying-in women. To Hofmeier and to Kaltenbach is due the credit of the independent discovery that the sugar was lactose. Spiegelberg regarded the presence of sugar as due to a "resorption diabetes." Ney, whose observations were more extensive than any before or since, embracing 24 pregnant and 148 lying-in women, also considered that the glycosuria was a form of "physiological diabetes" characteristic of good nurses. M'Cann and Turner carried out an admirable investigation on 100 cases, entailing 1400 analyses of the urine, and their results, which have lately been confirmed by Keim, may be taken as representing the present state of our knowledge on the subject. Lactosuria was observed in every case. As a rule, lactose was present every day after lactation. The quantity varied from ·18 to ·69 per cent, the average being ·35 per cent, *i.e.* 1½ grain to the ounce. The date of the first appearance of lactosuria was found to be as follows:—

1st day (day of labour)	29	per cent.
2nd ,,	27	,,
3rd ,,	26	,,
4th ,,	11	,,
5th ,,	2	,,

The late appearance of lactose on the fourth and fifth days can be explained by the fact that the supervention of lactation is delayed in these cases. M'Cann and Turner give two interesting tables to show the relation between the days when the largest amount of sugar is present and the day when milk first appears in the breasts. I have combined these tables to bring out the relation more forcibly.

	Percentage of cases when milk first appears in the breasts.	Percentage of cases when the largest amount of sugar is present.
1st day	—	1
2nd ,,	5	5
3rd ,,	10	46
4th ,,	26	39
5th ,,	26	6
6th ,,	8	1
7th ,,	5	
8th ,,	3	
9th ,,	4	
10th ,,	5	
11th ,,	3	
12th ,,	2	
13th ,,	2	
14th ,,	1	

Thus the mammary glands being in a state of great activity on the third and fourth days of the puerperium, milk is rapidly formed, and thus distension of the breasts soon occurs; at the same time milk-sugar is absorbed into the blood, owing to the excessive production or diminished outflow of milk, and this excess of milk-sugar is excreted by the urine, and is thus found in largest amount on the fourth and fifth days. In most cases, after this period, the amount of sugar remains constant if the milk be excreted uninterruptedly, and if the daily quantity be not excessive, *i.e.* if the production and exhaustion be equal.

The most important factor in causing increase of lactosuria is distension of the breasts, leading to absorption of milk-sugar into the blood. The same effect is produced by the application of belladonna to the breasts. Next in importance comes the excessive production of milk. But although excess of milk is associated with increased lactosuria, the quality of the milk may be inferior, except in regard to its richness in lactose. Hence the indication as to the value of a wet-nurse, which Blot thought could be found in the amount of sugar in the urine, is not to be relied on. Increased lactosuria also follows the reflexly increased secretion brought about by putting the child to the breast.

The Bladder.—Although an increased quantity of urine is secreted in the early days of the puerperium, the bladder usually displays a marked inactivity, with the result that retention of urine is generally met with. The principal reason of this is probably the same as is found operating in reference to the bowels, namely, diminished intra-abdominal pressure and laxity of the abdominal walls. A contributory cause is found in the bruised, sore condition of the bladder and urethra, for in the vesical portion of the latter fissures are often produced during labour (Spiegelberg). This acts in two ways: a reflex inhibition of the bladder muscle is brought about, and the patient dreads to pass water. Recht made observations on 224 women confined at term in the Clinique Baudelocque, and found that the time that elapsed between labour and the first micturition was as follows:—

Broadly expressed, the bladder was first emptied:

In the first six hours			in 51 cases,	or 22·76	%
From the 7th to the 12th hour			in 79 ,,	,, 35·26	%
,, 13th ,,	18th ,,		46 ,,	,, 20·54	%
,, 19th ,,	24th ,,		33 ,,	,, 14·75	%
,, 25th ,,	36th ,,		15 ,,	,, 6·69	%

The separate examination of primiparæ and multiparæ shows that retention is more pronounced in the former. The difference is probably largely accounted for by the greater extent and frequency of local injury or bruising.

	Primiparæ.	Multiparæ.
First micturition in first 12 hours	40·77 %	72·79 %
First micturition from 13th to 24th hour	48·54 %	23·97 %
First micturition from 25th to 36th hour	10·67 %	3·30 %

We shall return to the question from the point of view of treatment when discussing the management of the puerperal state.

5. *The Nervous System.*—For some hours after a normal delivery the patient lies very quiet; the mind is peaceful, owing to the feeling of satisfaction that labour is finished and the child is born. The physical condition resembles that of great fatigue, and after a prolonged labour it may even border on shock. Churchill thus describes it: "After a normal labour there is a condition of nervous shock which lasts for a few hours; during this time the sensibility of the brain is diminished, but the organs of sense are more sensitive. The eye has lost its brightness, and expresses languor and exhaustion; but it is more intolerant to light, as the ear is to sound. After obstetrical operations the shock may be much more pronounced." This passing phase of shock, if present, soon gives place to a condition of exalted irritability of the whole nervous system; so that very slight physical or emotional causes are sufficient to send up the temperature and otherwise disturb the patients. In those of a hysterical or highly strung temperament the border-line between sanity and insanity may easily be reached, and even overstepped. Normally, this condition subsides within a week or ten days.

6. *Lactation.*—For the first twenty-four hours after delivery the breast-secretion is much the same as in the later part of pregnancy, and is known as colostrum. The true milk-secretion begins most frequently on the third day. According to M'Cann and Turner's table, quoted above, the date of the first appearance of milk in the breasts presents the following relative frequency:—

1st day in	1 per cent	4th day in	39 per cent
2nd „	5 „	5th „	6 „
3rd „	46 „	6th „	1 „

Lactation is established rather later in primiparæ than in multiparæ. Its onset is marked by swelling and tenderness of the breasts; the superficial veins are distended, the axillary glands are enlarged, and the lymphatics leading to them are readily felt. The more pronounced these features are, the greater are the pain and the tenderness experienced; and when the local disturbances are considerable they are accompanied by general malaise, loss of appetite, and some rise of temperature. As we have remarked, the nervous system of the lying-in woman is excitable, and it is not therefore surprising that the temperature should show a slight rise when the breasts are very painful. This rise does not indicate that there is "milk-fever"; it is generally agreed now that no such thing exists. Moreover, as Griffith points out, it is important to remember that a rise of temperature which coincides with the onset of lactation may really be due to septic absorption from the genital canal, the effects of which appear about this time, and that disturbances in the breasts may actually be the result of such absorption.

At the outset, the secretion of milk, though independent of, is favoured by, the reflex stimulation of the gland through the nipple when the child is applied to the breast. Thereafter, as long as suckling is continued, the supply, under ordinary conditions, is kept up for nine or ten months. Sometimes, from constitutional causes, such as weakness, faulty diet or metabolism, or from no apparent cause, the supply runs short after three or six months; or, while the quantity is unchanged, the quality deteriorates, so that the child does not thrive. In other cases suckling is persisted in for twelve or fifteen months, and exceptionally for an even longer time. The weaning of the child is commonly followed within one or two weeks by diminution and disappearance of the milk. As long as lactation lasts, menstruation is usually in abeyance; but not infrequently the menses reappear at the fifth or sixth month, and occasionally a woman menstruates all through lactation. Conception during this period is correspondingly rare.

Characteristics of Colostrum and of Milk.—Colostrum is a turbid pale yellow fluid, alkaline, and slightly viscid from the presence of mucin. Chemically, it contains more fat, sugar, and inorganic salts than are found in the milk secreted later; and, in contradistinction to the latter, it contains a considerable quantity of globulin and very little caseinogen. Microscopically, it is characterised by fat globules, mucous corpuscles, shed epithelium, and colostrum corpuscles. The latter are swollen rounded cells loaded with fat globules; they are cast-off cells of the alveolar epithelium, and by suitable reagents can be shown to possess a nucleus, and to exhibit amœboid characters under certain conditions. Colostrum has a mild laxative action, due, according to Winckel, to its relatively large quantities of phosphate of calcium, and chlorides of sodium, potassium, and magnesium.

Human milk has a bluish-white tinge as compared with cow's milk, and, like the latter, has a neutral or faintly alkaline reaction. In carnivora the milk is acid. The chemical composition of human and of cow's milk is given by Frankland as follows:—

	Human Milk.	Cow's Milk.
Proteids (chiefly caseinogen)	2·7 per cent	4·2 per cent
Fat	3·5 „	3·8 „
Sugar (lactose)	5·0 „	3·8 „
Salts	0·2 „	0·7 „

Microscopically, milk presents the characteristics of an emulsion, with very finely divided fat globules and a few epithelial cells. Colostrum corpuscles are not found after the first few days.

Histological Changes.—Halliburton gives the following excellent account of these changes:—"The alveoli of the glands during the secreting periods are found to be lined with very short columnar cells, with nuclei situated towards the

centre. The edges of the cells towards the lumen may be irregular or jagged, and the remainder of the alveolus is filled up with the materials of the milk. During the intervals between the acts of discharge, the cells of the alveoli elongate towards the lumen, their nuclei divide, and in the part of the cells towards the lumen a collection of oil-globules and of other materials takes place. The next stage is that the cells divide, and the part of each towards the lumen containing a nucleus and the materials of the secretion disintegrates and goes to form the solid part of the milk. The cells also secrete water, salts, and milk-sugar. The fat, etc., of the milk are not simply picked out from the blood by the secreting cells, but these materials are formed by metabolic processes within the protoplasm of the cells. In the earlier days of lactation, epithelial cells only partially transformed are discharged in the secretion; these are termed "colostrum corpuscles."

7. *The Lochia.*—With the expulsion of the placenta a large raw surface is left at one part of the inner surface of the uterus, whilst a minor degree of denudation occurs over all the rest of this surface, owing to the detachment of the chorion. From the whole of this denuded area a discharge of blood and serum takes place; this discharge, mingled with shreddy fragments of decidua, and modified by the admixture of cervical and vaginal secretions, constitutes the lochia. The proportion of blood is greater than is found in the discharges from raw surfaces elsewhere, because the uterine vessels are not so perfectly secured as are those in an ordinary operation wound. In pre-antiseptic times the discharge usually became purulent after a few days, just as the formation of "laudable pus" was regarded as a normal stage in the healing of a wound; and consequently the lochia were distinguished, according to their character, as rubra, serosa, and alba. The total quantity of the discharges after labour was naturally greater when suppuration took place; and we accordingly find that the quantities which were formerly considered normal were greatly in excess of those met with at the present day. Gassner gave the following as the result of his researches:—

Lochia rubra, 1-3 day . .	1000 grms.
Lochia serosa, 4-5 day . .	280 ,,
Lochia alba, 6-8 day . .	205 ,,

Hence a woman after labour loses in 8 days 1485 grms. = 52·29 oz.

A nursing woman loses in all only 1085 grms. = 38·2 oz.

Whilst a woman not nursing loses 1880 grms. = 66·19 oz.

The majority of subsequent writers have quoted these figures, which, however, for the reasons given above, are not applicable to confinements conducted according to modern methods. Moreover, Gassner's observations were made on two cases only, one being a suckling and the other a non-suckling woman. In order to determine the question of the normal amount of the lochia, I carried out an investigation on 64 consecutive cases at the General Lying-in Hospital, Lambeth. The method of procedure was as follows:—

The lochia were received in wood-wool pads covered with sublimate gauze. These pads were made carefully to scale in two sizes, 1 oz. and ½ oz. respectively; the former being employed at first in each case, and replaced by the smaller ones as the discharge diminished.

After removal, each pad was at once again weighed, the difference representing the amount of discharge during the time that the pad was on.

When, two or three times in succession, the soiled pad was found not to have gained in weight, the lochia were regarded as having ceased.

Four of the cases were not normal, and were not included in my calculations. The remaining 60 cases gave the following results:—

The lowest quantity was 2 oz., the highest 24 oz.

In 30 cases, or 50 per cent, the quantity was 10 oz. or less.

In 19 cases, or 32 per cent, the quantity was 10-15 oz.

In 8 cases, or 13 per cent, the quantity was 15-20 oz.

In 3 cases, or 5 per cent, the quantity was over 20 oz.

The average quantity, taking the whole of the 60 cases together, is 10·89 oz. If we exclude three cases above 20 oz., the average, calculated on 95 per cent, is 10·2 oz. Hence we conclude that the average normal quantity of lochia is about 10½ oz.

The duration of the discharge was from five days to a fortnight, and was distributed as follows:—

In 4 cases .	5 days	In 6 cases .	10 days
In 4 ,, .	6 ,,	In 7 ,, .	11 ,,
In 10 ,, .	7 ,,	In 6 ,, .	12 ,,
In 11 ,, .	8 ,,	In 1 ,, .	14 ,,
In 11 ,, .	9 ,,		

That is, in 40 cases, or 66 per cent, it was nine days or under. The average on all the cases is 9·75 days.

A practical deduction is that it is usually safe for the patient to get up on the tenth day.

As to the factors which may influence the quantity of the lochia, the chief interest, in view of Gassner's results, lies in the question of suckling. On this point my cases do not bear out Gassner's conclusion that the quantity is considerably increased in non-suckling women.

In 53 cases in which the patient nursed the baby the average was 11·2 oz.; in 7 cases in

which the child died, or was fed by hand, the average was 10·3 oz. The number in the latter class is small, as only two were still-born, and among the others weaning was not encouraged. But though the number is too small for much stress to be laid on the figures, it is at least clear that non-suckling does not increase the quantity of lochia.

The age and parity of the patient were found to bear no relationship to the amount of the lochia. On the other hand, the quantity was found to vary directly with the weight of the child, and especially with that of the placenta; with the amount of blood lost at the time of labour, and with the habitual amount of the menstrual flow; whilst the quantity was also greater in the case of dark colouring and complexion than with fair women.

B. *Changes in the Pelvic Organs—Involution.* —1. *The Uterus* —The changes in the uterus constitute by far the largest and most important feature of the process of involution. For whereas we have to study, in the abdominal wall, the recovery from distension; in the uterine appendages, broad ligaments, and bladder, the return from mechanical displacement; in the vagina, vulva, and perineum, the recovery from violence during labour; we have to follow, in the case of the uterus, the extraordinary and rapid changes by which an organ weighing 2 to 3 lbs. is transformed, within six or eight weeks, into one weighing 1 to 2 ounces. The process is rapid enough to be readily traced at the bedside, and we shall take this aspect of the subject first; after which will be considered the anatomical changes on which the clinical phenomena are based. Lastly, we shall have to inquire how far the way in which involution is effected is explained by histological and chemical data.

(*a*) *Clinical Features.*—At the conclusion of the third stage of labour the uterus can be felt, under normal conditions, firmly contracted, and lying in the hypogastrium, reaching nearly up to the umbilicus. It has been aptly compared to a cricket-ball, in the impression it conveys to the hand. Contraction is not continuous, but rhythmical; but in the intervals of relaxation it should remain retracted, and it is found in this condition except when hæmorrhage is going on or when distended with clots. On palpating the uterus during the phase of retraction with relaxation, it feels rather soft and ill-defined, and its upper border usually reaches a slightly higher level than during contraction. With the progress of involution the uterus gradually recedes from the abdomen, and when the process is complete it has resumed its normal non-parous position of an entirely pelvic organ. By noting the position of the uterus on the successive days of the puerperium we can obtain a valuable clinical indication of the progress of involution, or, on the other hand, of its disturbance and interruption by pathological conditions, of which we thus receive an early warning.

The estimation of the uterine recession has been attempted in various ways. Hecker, Buhl, and Scanzoni measured the uterus of women who died at different times after delivery. Serdukoff collected from the tables of these authors the cases where death did not apparently arise from disease of the sexual organs, and where, consequently, involution was almost normal; and he thus got 23 cases, including 2 of his own. But he concluded that no reliable data could be derived from such a small number. Serdukoff also attempted to gauge the uterine measurements by the introduction of the sound; but he concluded, as other writers have done, that this plan, besides being not devoid of risk, was also unreliable in its results. Sinclair adopted the sound for measuring the uterine cavity at various intervals after delivery; but, as only one observation was made on each of his 324 cases, his results are of little value for the present purpose.

Wieland was the first to adopt the plan of external measurements of the length and breadth of the uterus. He concluded that up to the seventh day there was a daily diminution in length of from 1 to 1½ cm., and after that of ½ to 1 cm. At the tenth day he described the uterus as being out of reach of measurement, having receded behind the symphysis. Schneider carried out some observations by a similar method. Serdukoff undertook a careful investigation, with special precautions to exclude sources of error; he concluded that the transverse measurements gave a more reliable indication of involution than the vertical ones. We shall have to refer later on to his *résumé* of the circumstances by which involution is affected.

The best recent work is that of Stevens and Griffith. They advocate vertical measurements, because the transverse measurement is difficult to take, and not practicable with any degree of accuracy after the first four days, unless the uterus remains abnormally large. To obviate fallacies, the measurement must be taken at nearly the same hour daily; the bladder and rectum must be empty, and the uterus must be in the normal mesial position and anteverted, and must be measured in a state of contraction. The character of the pelvis must also be borne in mind: in a flattened pelvis the uterus will be found considerably higher than in a kyphotic pelvis (Barbour). Stevens and Griffith suggest a convenient way of recording the variations in the height of the fundus, by the use of the ordinary temperature chart; the 100° F. line of the chart is taken to represent the pubes, and the lines above, 101°, 102°, etc., represent in inches the height of the fundus above the pubes.

The height of the fundus on successive days is here given according to Stevens and Griffith (the figures representing approximately the

mean of the morning and evening records) and Luisstag.

	Stevens and Griffith.	Luisstag.
1st day	$5\frac{1}{4}$ inches	6 inches
2nd ,,	5 ,,	$4\frac{4}{5}$-$5\frac{1}{5}$,,
3rd ,,	$4\frac{2}{3}$,,	$4\frac{2}{5}$,,
4th ,,	$4\frac{1}{3}$,,	4 ,,
5th ,,	4 ,,	$3\frac{3}{5}$,,
6th ,,	$3\frac{1}{2}$,,	$3\frac{2}{5}$,,
7th ,,	$3\frac{1}{3}$,,	$3\frac{1}{5}$,,
8th ,,	$3\frac{1}{6}$,,	$2\frac{4}{5}$,,
9th ,,	$2\frac{5}{6}$,,	$2\frac{3}{5}$,,
10th ,,	$2\frac{3}{4}$,,	$2\frac{2}{5}$,,
11th ,,	$2\frac{1}{3}$,,	$2\frac{1}{5}$,,
12th ,,	$2\frac{1}{6}$,,	2 or under.

Varnier thinks that Luisstag's figures are too high, and says that he has often found the height of the fundus less than 5 inches in the second half of the first day; 4 inches on the second day; $2\frac{2}{5}$ to $3\frac{1}{5}$ at the fifth to seventh day; and lying behind the pubes towards the ninth day.

The height of the fundus is increased by

(1) Distension of bladder, rectum, and intestines.

(2) Mechanical distension of the uterus by clots, retained placenta, or membranes.

(3) Sub-involution.

(4) Anatomical peculiarities, such as abnormal high position of the uterus or unusual thickness of its walls.

The height of the fundus is diminished by malposition of the uterus—namely, prolapse, retroversion, extreme anteflexion, and excessive lateral obliquity.

In cases in which there is a doubt as to whether the enlargement of the uterus, as indicated by the height above the symphysis, is apparent or real, the external measurement should be checked by a bimanual examination, which, though of little value as a precise measurement, will give a fairly accurate indication as to increase in size.

(*b*) *Anatomical Features.*—Our knowledge of the anatomy of the uterus during the puerperium is mainly based on observations of frozen sections made by the following authorities :—

1. Stratz (1886), I-para, 1 hour after delivery. 2. Barbour and Webster (1891), multipara, immediately after delivery. 3. Barbour (1888), I-para, $1\frac{1}{2}$ hours after delivery. 4. Webster (1892), multipara, 2nd day after delivery. 5. Webster (1892), multipara, 3rd day after delivery. 6. Varnier (1900), (?), 3rd day after delivery. 7. Varnier (1900), (?), 3rd day after delivery. 8. Webster (1892), multipara, $4\frac{1}{2}$ days after delivery. 9. Barbour (1888), I-para, $5\frac{1}{2}$ days after delivery. 10. Webster (1892), multipara, 6 days after delivery. 11. Webster (1892), multipara, 15 days after delivery.

The following description is based on the work of these authors.

i. *Macroscopical Characters.* — Immediately after delivery the uterus consists of a firmer thick-walled upper portion and a lower flaccid thin-walled portion. The transition between the two portions is sharply defined on the anterior wall, but more gradual on the posterior wall. The line of union of the upper and lower portions represents a retraction ring, which is not that of labour, but is nearer the cervix, part of the lower segment having been taken up into it. The cervix is thick, flattened vertically, and partly everted; the os internum cannot be defined, even microscopically. The whole organ measures about 8 inches in length and $3\frac{1}{2}$ inches antero-posteriorly; the cavity measures 7 inches. The weight is about $3\frac{1}{4}$ lbs. (Varnier). The softness of the lower segment causes it to lie in a somewhat crumpled-up condition; the whole uterus is anteverted, but not anteflexed. The thickness of the walls of the upper segment is about $1\frac{1}{2}$ inches anteriorly and posteriorly, and about 1 inch at the fundus; in the flaccid portion the anterior wall is about $\frac{1}{2}$ an inch and the posterior $\frac{3}{4}$ of an inch in thickness. The peritoneum has a firm attachment on the posterior aspect of the uterus; but anteriorly it can be stripped up to a "line of firm attachment" which has a position varying from 2 to 4·8 inches above the anterior lip (Barbour).

When the uterus is laid open it presents a pear-shaped inner surface, which consists above of an oval area 7 inches traversely and $5\frac{1}{2}$ inches from above down. This passes below into a band 4 inches wide and 2 inches vertically. The band is the opened-out cervix; it is of a bluish-red colour (due to ecchymoses) and comparatively smooth, save for the branches and stem of the arbor vitæ. The oval upper area consists of two parts—a ragged and dark purple patch, about one-sixth of the whole in size, surrounded by a brick-red surface mottled with numerous tags. The former is the placental site, the latter the decidua-covered part of the uterus (Barbour).

The uterus is still abdominal on the fourth day, and on the sixth is just within the brim at the conjugate. The vertical measurements are thus summarised :—

Cases.	Cervix.	Body.	Whole Uterus.	Cavity.
2nd day	$2\frac{1}{8}$ inch	$4\frac{7}{8}$ inch	$7\frac{1}{8}$ inch	$6\frac{1}{8}$ inch
3rd ,,	2 ,,	$4\frac{3}{8}$,,	$6\frac{3}{8}$,,	$5\frac{5}{8}$,,
4th ,,	2 ,,	$5\frac{1}{2}$,,	$7\frac{1}{8}$,,	$6\frac{1}{4}$,,
6th ,,	$1\frac{5}{8}$,,	$3\frac{1}{2}$,,	$5\frac{1}{8}$,,	$4\frac{5}{8}$,,
15th ,,	$1\frac{1}{4}$,,	$2\frac{1}{2}$,,	$3\frac{3}{4}$,,	$3\frac{3}{8}$,,

There is but little diminution in size for the first four days. On section there is the appearance of contracted and anæmic non-striped muscle, without any naked-eye appearance of fatty degeneration. The post-partum uterus acts as a plug in the pelvis. The ovarian and uterine arteries are twisted and compressed by the uterus against the pelvic wall; the uterus

with the exception of the cervix, being anæmic. The cervix is congested and will bleed easily; post-partum hæmorrhage from the torn cervix is most marked where the pelvis is large (because the uterus does not plug so efficiently) or contracted (because the uterus cannot sink down).

The cervix, which at the end of labour is soft and limp, and which passes insensibly into the body of the uterus, gradually resumes its normal characteristics within the first fourteen days; by this time the arbor vitæ shows its characteristic folds, the canal has narrowed, and the internal os is readily distinguished. By the twenty-first day the canal has contracted to the normal calibre, and the internal os is sharply defined. At the end of the sixth week the cervix is quite normal.

ii. *Microscopical Characters.*—Wm. Hunter taught that the post-partum uterus is lined by a "stratum" of decidual membrane; Cruveilhier and Heschl, in later times, held the view that the uterus was laid bare down to the muscle. Matthews Duncan supported Hunter's view, which has been confirmed by the work of Friedländer, Kundrat and Engelmann, Langhans, Leopold, Küstner, Turner, and others. This stratum is about 2 mm. in thickness, and consists of uterine glands imbedded in interglandular tissue. The glands are lined, at their deeper part, by a cubical epithelium; in the middle portion, where the dilatation of the glands gives the appearance which has led to the designation of "the spongy layer," the epithelium is often wanting. In many cases the line of cleavage in the separation of the membranes appears to have passed through this layer; but in other cases a more superficial and compact portion of the glands remains, and here the epithelium is again found, of a columnar type. A surface epithelium also is sometimes present. In the portion of the decidual area nearest the cervix the mucosa is generally found in a more complete form than in the area nearer the placental site.

The mode of involution of the uterine muscle was also formerly a disputed point. Kilian (1849), who believed that the hypertrophy of the uterus during pregnancy was due both to enlargement of existing muscle fibres and to the new formation of others, held that both kinds of muscle-fibre disappeared completely by a process of fatty degeneration. Heschl supported this view of involution, saying that, of the uterus which existed before labour, not a fibre survived. Kölliker also attributed involution to fatty degeneration, but thought that most of the muscle-fibres remained, though they diminished in length and thickness. Robin (1848-1861) held a quite different view, maintaining that the process was one of simple atrophy without fatty degeneration, and that none of the muscle-fibres disappeared. Meola (1885) attributed the regression of the muscle-fibres to a process of cirrhosis due to a rapid proliferation of the intra-muscular fibrous tissue. Sänger (1888) denied the proliferation of the connective tissue, but supported the doctrine of fatty degeneration, without, however, any disappearance of muscular fibres. Mayor's (1888) explanation was very similar to Sänger's. Helme (1889), in a careful investigation, revised the question, and his conclusions are now accepted as final. He finds that involution "does not bring about a destruction of the cells. It is merely a transition from larger to smaller dimensions. . . . It is an incomplete atrophy—not a numerical, but a simple physiological atrophy; the process in each fibre goes on to a certain point and then stops—the fibre remains, only it is of smaller volume." As to the way in which this is brought about, Helme finds no evidence whatever of fatty degeneration; neither is there any increase of connective tissue. "The change is one of physiological retrogression; the actual chemical character is not revealed by the microscope, but it is probable that the cell-protoplasm undergoes a sort of peptonisation—all the surplus material that is no longer required being changed into some more soluble substance, which finds its way into the surrounding lymph." Helme's explanation of the process receives some support from Fischel's researches, according to which peptonuria is a constant feature of the puerperium, whilst peptone is also found in the lochia and in the uterine muscle. Helme found that there was a gradual and progressive diminution in amount and disappearance of the connective tissue. Some of the connective tissue fibres during the first three days swell up and become hyaline, eventually breaking down into granules. The connective tissue cells also become more and more granular, and their nuclei are greatly changed, breaking up into fragments. In some places the destruction is so complete that all traces of connective tissue fibres are lost, their place being taken by a clear matrix containing a mass of entangling granules and degenerating leucocytes, and corpuscles and nuclei. Scattered through this are the large plasmodia, which begin to be formed in the later days of pregnancy, and whose function is evidently to eat up the waste material lying around them. At the same time, large granular epithelioid cells are found, with a single clear vesicular nucleus; on the sixth day they are found to be almost filled with bright yellow granules derived from the disintegrating red blood-corpuscles.

In both arteries and veins there is observed in the smaller vessels a process, first of compression, then of obliteration; whilst many of the larger vessels exhibit a proliferation of the intima, which results in a complete blocking of the lumen, and the conversion of the vessel into a fibrous cord which ultimately disap-

pears. The plasmodia are especially congregated round the vessels, and no doubt they assist in absorbing the contents of the cells of the vessels which are undergoing the gradual diminution.

2. *The Uterine Appendages and Broad Ligaments.*—The outer or pelvic extremity of the broad ligaments and Fallopian tubes being relatively fixed, it follows that, as the uterus enlarges during pregnancy and rises in the abdomen, the tubes come to lie more or less parallel to the vertical axis of the uterus; and this is also the position in which they are found during the early part of the puerperium. The greater proximity to the uterus which results, and in which the ovaries take part, is further accentuated by the lateral expansion of the lower part of the uterus, which encroaches on the cellular tissue of the lower part of the broad ligaments and separates their anterior and posterior surfaces. The ovarian ligaments become temporarily obliterated, being lost on the lateral surface of the uterus. As the uterus becomes progressively smaller, the layers of each broad ligament fall together, and the Fallopian tube and ovary become carried away from the side of the uterus, till, when involution is completed, the former resumes its transverse direction. The tubes are generally lengthened as well as drawn up during the pregnancy, and the same remark of course applies to the upper border of the broad ligament. If the involution of these structures does not keep pace with that of the uterus, the tube and ligament come to lie rather slack, with the result that the ovaries and tubes easily become prolapsed into the pouch of Douglas. The elongated round ligaments of the uterus also shorten during the puerperium; if they fail to do so rapidly enough, or to a sufficient degree, there is a great predisposition to backward displacement of the uterus.

3. *The Vagina.*—The vaginal walls, enlarged both in width and in length, remain lax and flaccid at first, but the sigmoid shape of the vaginal canal may be restored in three days (Webster). The anterior and posterior walls of the vagina fall into apposition almost at once in their upper portion; the lower portion, however, gapes. The posterior wall, being relatively fixed in its upper portion, is generally but little disturbed, except from such laceration as may have occurred in its lower part, and by efficient repair of perineal tears all bad results are avoided. But with the anterior wall it is otherwise. The attachment to the back of the pubes through the medium of the base of the bladder is generally so far loosened that there is nearly always a tendency to some prolapse in the form of cystocele, and this may persist, especially when labours succeed each other rapidly.

The pelvic-floor projection is increased for the first few days. Thus Webster found the measurements in his cases to be as follows:—First day, 2 in.; second, $1\frac{7}{8}$ in.; third, $1\frac{5}{8}$ in.; fourth, $2\frac{1}{8}$ in.; sixth, $1\frac{1}{2}$ in.; and on the fifteenth day, 1 in.

4. *The Bladder.*—After delivery the bladder resumes its normal shape, and when empty it lies below the level of the symphysis pubis. Halliday Croom stated that the bladder may be higher after delivery than it was before. Webster, however, from the study of his frozen sections, considers that this view is wrong, and that the bladder is never higher, but may be lower during the lying-in. After severe or repeated labours the base of the bladder is partially loosened from its pubic attachments, and may bulge down and backwards, in conjunction with the anterior vaginal wall.

A considerable measure of the interest which centres round the puerperal bladder is derived from the influence which a full bladder exerts on the height and position of the uterus. The question has been studied in great detail by Croom. According to him, distension of the bladder pushes the uterus up higher and away from the abdominal wall, and also causes some deviation to one side or other. It has been generally taught that this deviation is to the right, but Croom considers that left lateral deviation is more common. The main reason for this is that, as the bladder fills, it generally expands to the right. When the uterine deviation is to the right, he regards it as due to a previous right deviation during pregnancy, to loading of the rectum, or to accidents of position. There is usually some asymmetry of the bladder, even in the non-puerperal condition. According to Barkow, of 35 bladders in the adult female, 4 were symmetrical; there was a minor degree of asymmetry in 10, major in 21. The deviation was to the right in 18 cases, to the left in 8. In those cases where there is rotation of the uterus on its longitudinal axis, the rotation is emphasised by a full bladder. Croom found that there was rotation in 10 out of 40 cases examined. This corresponds with the observation of Börner, who found that out of 64 cases there was no rotation in 50; rotation to the right in 12, and to the left in 2. Haig Ferguson believed that there might be sufficient rotation to bring the left ovary so far forward as to be grasped in the Crédé manipulation. According to Webster, the rotation of the uterus is insufficient to bring its transverse diameter into the right oblique diameter of the pelvis, and the ovaries do not take up positions corresponding to the rotation. As to the amount of urine which suffices to cause upward and lateral displacement of the uterus, Pfannkuch says that 100 c.c. (3·5 oz.) of urine raise the uterus about 1 cm. ($\frac{2}{5}$ in.). Autefage states that 400-600 grammes (14 to 21 oz.) of urine will cause an increase in the real height

of the uterus which is not less than 4 to 9 cm. ($1\frac{1}{2}$ to $3\frac{1}{2}$ in.). Croom attributes these results to observations being made at varying periods of the puerperium. He finds that in the early puerperium the uterus is not displaced appreciably till the bladder contains 20 to 30 oz. of urine.

5. *The Abdominal Walls.*—After the great distension produced by the pregnant uterus, the abdominal walls after delivery lie flaccid and loose, appearing to be too large for the abdominal contents, and the skin is wrinkled. For some weeks the muscles are lacking in tonicity, and, owing to their elongation, they cannot contract efficiently. To this cause among others may be traced the paresis of the vaso-motor system, of the bowels, and of the bladder. In some cases, and especially after rapidly succeeding pregnancies, the common tendon of the internal oblique and transversalis is so stretched that the two recti remain separated, and when the patient strains the abdominal contents bulge between them, causing a ventral hernia in the form of a long, low vertical ridge. When the recti and the lateral abdominal muscles alike fail to regain their tone a pendulous belly results, which is not only inconvenient and ungraceful at ordinary times, but may result in considerable inconvenience during subsequent pregnancies, leading to malpresentations and to inertia during labour.

6. *Evidences of Recent Delivery.*—The diagnosis of recent delivery has sometimes a medico-legal importance, and it may depend on anatomical or on clinical signs. Anatomically, the condition of the uterus is of chief importance. It is not necessary to recapitulate what has already been said on this point.

Clinically, evidence is derived from the condition of the abdomen, breasts, and genital tract. On examining the abdomen its walls are loose and ill-fitting, and traversed by striæ of a more or less vivid red. It is assumed that the rapid emptying of the abdomen due to other causes, such as the evacuation of fluids or the removal of tumours, can be excluded, for in this manner also the lax condition of the abdominal walls could be accounted for. The striæ, however, are seldom so well marked or so red in other conditions as they are in the puerperium; and moreover, there is usually a marked pigmentation of the linea alba after labour. On palpation of the abdomen, the enlarged uterus can be felt occupying the pelvis. Further examination will be required to distinguish this from other kinds of tumour. The breasts are enlarged and tense, and present a large dark areola. Milk is freely secreted. The condition of the breasts gives but little indication as to the date of the delivery, except that, if the secretion consists of colostrum, labour must have occurred only one or two days previously.

On examination of the genital tract, lochia are found flowing from the vagina. If they are mainly composed of blood, we have probably to do with the first ten days of the puerperium; later, the discharge is sero-sanguinolent, and, later still, serous. The vulva is found swollen, and appears rather bruised. Some laceration of the fourchette or perineum will usually be present. The vagina is lax, stretched, and may present some laccrations, whilst its lower portion gapes. The cervix is soft and thickened, and for the first day or two after labour its outlines may be difficult to define. The lips of the cervix present some recent lacerations or fissures, and the canal is patulous, especially in its lower portion, where for the first few days it readily admits one or two fingers. Bimanually, the uterus is felt to be enlarged and usually strongly anteverted. The date of delivery can be roughly estimated by the height of the fundus uteri above the symphysis pubis. If the case be watched for a week or ten days, the gradual diminution in size of the uterus is quite characteristic, as it can be simulated by no other condition.

II. The Management of the Puerperium

1. *First Duties.*—After the expulsion of the placenta the patient should be cleansed and made comfortable. The advisability of a vaginal douche as a matter of routine is the subject of some difference of opinion. In hospital practice we think that the douche should always be given, whilst in private practice it is sometimes better that it should be omitted. The indications for the employment or withholding of the douche may be broadly formulated as follows :—

The douche is indicated (*a*) in all cases of operative delivery, such as forceps, turning (by internal manipulations), and craniotomy; (*b*) in other cases where the hand has been introduced, as for the replacement of the funis, the bringing down of the arms in extension above the after-coming head, or the extraction of placenta or membranes; (*c*) in tedious or difficult labours, involving injury to cervix, vagina, or perineum; (*d*) when the fœtus is decomposed; (*e*) when purulent or muco-purulent discharges were present before labour; (*f*) when there is a tendency to post-partum hæmorrhage.

The douche may be withheld (*a*) when labour has throughout been spontaneous, and no vaginal examination has been made; (*b*) when a few examinations have been made in a normal case, and the uterus contracts well.

An intra-uterine douche should be given only when there has been intra-uterine manipulation, or when the uterus does not contract well.

After the cleansing a binder should be applied. It should consist of a material sufficiently soft to allow of its fitting closely; below, it should reach four inches below the trochanters, and above it should just come up to the front of the

costal margin. The binder is supposed to fulfil three objects: first, to give the patient a sense of comfort; second, to counteract the diminished intra-abdominal pressure; third, to restore the patient's "figure." From measurements carried out on three series of patients, the first with the binder applied in the ordinary way, the second without binder, and the third with a binder applied and kept specially firm, Herman came to the conclusion that, considered as a means of keeping the waist small, a binder of the ordinary kind has not the slightest effect. Nor has a binder any effect in causing or maintaining uterine contraction. In the discussion on Herman's paper at the London Obstetrical Society, Champneys expressed the view that the absence of a binder favoured the production of the condition of pendulous belly, and that the use of a binder was to keep the abdominal walls in a favourable condition and position during the involution which they, in common with other structures hypertrophied during pregnancy, should undergo during the lying-in period.

When the patient has been made comfortable, the bed should be made, a draw-sheet being placed under her. The room should be darkened and left with a window open, and the patient should be encouraged to sleep. The accoucheur should not leave the house till the pulse has become lower than 100, this being a sign that there is no special tendency to hæmorrhage. After a few hours the baby, which has meanwhile been washed and dressed, may be put to the breast for a few minutes. The nurse should be instructed to keep a careful watch for any symptoms of hæmorrhage, by looking at the pads and by feeling the abdomen from time to time, to ascertain that the uterus is contracted or retracted. At the first subsequent visit the obstetrician should, as a matter of routine, inquire about after-pains and the emptying of the bladder; the pulse and temperature should be noted, as well as the patient's general condition; he should see the pads and make special inquiries as to the passing of clots; and lastly, he should examine the abdomen, to ascertain the condition of the uterus and of the bladder. The latter will sometimes be found distended, even when the patient is alleged to have passed water.

After-pains are due to painful uterine contractions, and are usually most pronounced when the uterus has not become exhausted by a difficult or tedious labour; they are, therefore, more common in multiparæ than in primiparæ. They also become more severe when clots require to be expelled; and they may be induced reflexly by the application of the child to the breast, or directly by the application of the hand to the abdomen. When they occur in moderation they are rather a welcome sign than otherwise. But if they become excessive they are bad, both because they are disturbing to the patient, and also because they may then be due to some irritation of the uterus, or to a pathological condition within its cavity. When, therefore, after-pains are moderate, they require no treatment; but when they are severe they must be relieved, and they may also constitute an indication for intra-uterine interference. The latter is specially indicated if the uterus is found to be bulky, or lying too high in the abdomen. For the relief of after-pains, in most cases, nothing is so effectual as phenacetin; it may be given in a dose of 10 to 15 grains, repeated, if necessary, in one or two hours. In severer cases, chloral may be administered in doses of 20 to 30 grains. Opium and morphia should only be given as a last resource.

During the whole of the puerperium, the pulse and temperature should be regularly recorded; and it is a great advantage for the daily height of the fundus to be recorded at the same time, in the manner suggested by Stevens and Griffith. The chart will then constitute a record of progress which will be of special value, since any unfavourable indications can be detected at a glance.

2. *Cleanliness* is perhaps the most important individual factor in securing a favourable convalescence. The patient's body-linen and bed-linen should be kept scrupulously clean, and changed as often as necessary. The external genitals should be washed several times a day for the first few days, and the vulvar pads should be renewed every time. In normal cases daily douching is not necessary; it is quite sufficient that the outside parts be washed and carefully dried. Any offensiveness in the discharges should, however, be the signal for a vaginal douche; for this purpose, biniodide of mercury (1 in 3000), iodine (1 drachm of the tincture or liniment to the pint), sanitas (1 part to 5 of water), or chinosol (1 in 3000) may be used. An intra-uterine douche should not be given except by the medical attendant. The patient should be sponged daily with tepid water, from head to foot, and carefully dried; only a small part of the patient's body should be exposed at a time. The breasts require special attention, so that they may be kept clean, and free from milk becoming sour.

3. *Diet.*—Formerly a low diet was considered the proper one for the lying-in woman. So long ago as 1864, Graily Hewitt uttered a protest against this practice, urging that the diet should be a generous one, both because the patient's strength required building up, and because she had to nourish the baby as well as herself. At the present time a rational treatment more largely prevails. For the first twenty-four hours the diet should consist of liquids, but it should be nourishing, and the "tea and toast" tenets should be relegated to the same limbo of defunct prescriptions as the "starve a fever" principle.

In a normal case, if the patient is hungry at the conclusion of labour, she may have warm milk, soup, gruel, or cocoa. In other cases, she is better left undisturbed in every way till after her sleep, when the same kind of nourishment should be given. On the second day she may take such things as custards, soft-boiled eggs, corn-flour, milk-porridge, etc. From the third day onwards a light ordinary diet is indicated, including white fish, chicken, lamb or mutton, merging into the diet of daily life. For particular cases, certain modifications are necessary. Thus, after prolonged anæsthesia, no nourishment should be given till its effects have passed off; in fact the case must be treated as a post-operative one. If febrile symptoms develop, a liquid diet will have to be maintained for a longer time. If the patient does not nurse the baby, the diet will not require to be so full as when nursing. Stimulants are best avoided in ordinary cases. The popular superstition that stout has specific virtues in the formation of milk must go the way of all superstitions; the best drink for the lying-in woman to take with her meals is good milk.

4. *Regulation of the Bladder and Bowels.*—As has been already mentioned, there is a considerable tendency to retention of urine after labour. This may act injuriously on the bladder itself, and favours post-partum hæmorrhage by the tendency to uterine relaxation which it induces. The patient should therefore be encouraged to empty the bladder within eight or ten hours after delivery; and even if she micturates spontaneously, the doctor or nurse should ascertain, by abdominal palpation, that the bladder is really emptied. There is some divergence of opinion as to the advisability of using the catheter when the patient cannot pass water alone. Varnier considers that it does no harm for the patient to go for twenty-four hours, and that the risk of cystitis precludes the use of the catheter in all but exceptional cases. The opposite view, which I emphatically endorse, is that at the end of twelve hours, if fomentations or change of position have a negative result, the catheter should be passed. If a glass catheter be used, it can be boiled every time after use and kept perfectly clean, and there is then no risk at all of cystitis. When the perineum has been torn and sutured, it is better to pass the catheter from the first, and to continue its use for thirty-six hours, to avoid contamination of the wound. It is of the greatest possible importance that when the catheter is used, the vulva should be carefully cleansed with warm water before the introduction of the instrument, and that the catheter should be introduced in full view, without fumbling. It is courting disaster to employ the old plan of feeling under the clothes, sweeping the finger over anus and vagina to the urethral orifice, and then passing the catheter along a contaminated finger; and the inconsistency of passing a carefully sterilised catheter into a urethra whose orifice lies amid unhealthy discharges is an absurdity that needs no demonstration.

Evacuation of the Bowels.—If the bowels have been carefully regulated during the latter part of pregnancy, and the rectum emptied by an enema during the first stage of labour, the obstetric field will be clean during delivery, and the daily action of the bowels will probably continue with very little interruption during the puerperium, especially if a proper diet has been given from the first.

The old plan of leaving the bowels locked up till the third or fourth day is becoming superseded by the practice of securing an evacuation within the first thirty-six hours, if there is not a spontaneous action. Castor-oil is one of the most effective aperients; but besides its nauseous taste, it sometimes causes griping; and in general the patient may be given the aperient to which she is most accustomed; or a saline laxative such as citrate of magnesia may be administered, followed, if necessary, by a simple enema, or a glycerine injection or suppository. Thereafter a daily evacuation should be secured.

5. *Nursing and the Care of the Breasts.*—Every healthy woman should nurse her own baby, unless important reasons forbid it, both in her own interests and in those of the child; for lactation favours a healthy involution, and the child has always the best chance of thriving when brought up at the breast. Nursing is contra-indicated when the mother is the subject of chronic exhausting disease, especially tuberculosis; in the majority of acute diseases; when syphilis has been acquired late in pregnancy; when the supply of milk is insufficient; when the breasts are inflamed; in many cases when the nipples are under-developed, inverted, depressed, persistently sensitive or sore. In some cases of the latter class the use of a nipple shield will allow the continuance of the function with comfort to the mother and with advantage to the child.

The baby should be first put to the breast six to eight hours after labour, and then every four hours during the first few days. Regularity should be practised from the beginning; it is harmful to put the child to the breast every time it cries. Both breasts should be equally used. Sometimes, when the supply of milk is rather short, it is a good plan, when the baby is put to one breast, to place a receiver to the other; for the stimulation of one nipple usually causes reflexly a flow from both breasts at once, and the milk so collected can be kept warm and used to supplement what the child obtains by suckling. If, on the other hand, the breasts tend to become over-distended, some milk should be drawn off with the breast-pump; for when much distended, the nipple is relatively shortened;

the child cannot get hold of it properly, and presses its nose against the breast, blocking the nostrils; it then has to let go the nipple in order to breathe. The frequent grasping and letting go discourages the child, produces soreness of the nipples, and leaves the breast insufficiently emptied. (*See also* "Affections of Breast and Nipples," p. 369.)

6. *Examination of the Pelvic Organs.*—Inasmuch as many of the diseases of women are due to overlooked accidents occurring at the time of labour, and to unfavourable circumstances during the lying-in period, including too early a return to daily duties, it is important that an examination of the pelvic organs should be made before the patient is allowed to leave her bed. By the tenth or twelfth day in normal cases the vaginal outlet has nearly regained its normal dimensions, and there is no appreciable deficiency of the perineum. The lochia have practically ceased, although there may be a little return when the patient begins to sit up. The cervix is firm, its canal is contracted, and there are no deep lacerations. The uterus is movable, lies in a normal position, or is slightly "squatting" back, and is only a little above normal size, the sound entering for about 3 inches. The region of the broad ligaments is free of any effusion, and the ovaries and tubes can be felt, normal in size and position.

7. *Getting up and going out.*—It is advisable, in a normal case, that the patient should be allowed to occasionally sit propped up in bed at a fairly early stage of the puerperium, namely, from the fourth or fifth day; thus she may be allowed to sit up a little for her meals. The change of posture favours uterine and vaginal drainage. If an examination shows that the pelvic organs are in the healthy condition described in the previous paragraph, the patient may be transferred to a couch as early as the tenth day; it is, however, an advantage for her to remain in bed for a full fortnight. Getting up must be postponed when the involution of the uterus is not satisfactory, when the lochia remain abundant, or when a torn perineum has failed to heal properly; whilst any effusion in the cellular tissue by the sides or in front of the uterus will necessitate a delay of some weeks. Undue laxity of the abdominal walls requires treatment at this early stage. Whatever the effect of the binder, a pendulous belly or a condition of wide separation of the recti is due to enfeebled abdominal muscles; and nothing has such a good effect, both immediate and remote, on these conditions as the proper exercise of the muscles. After the first ten days the patient should be instructed to practise lying flat on her back, and raising herself into a sitting posture without using her hands and arms. This should be done for five or ten minutes at a time twice a day.

Once the patient has begun to get up, a return to the ordinary mode of life should be effected in easy stages. After a couple of days on a couch, she may sit up in an easy-chair, and begin to walk a little. A few days more, and she may go into another room, downstairs if necessary. Then a fine morning or afternoon is made the opportunity for a drive; and lastly, she may walk out at the end of the third week or the beginning of the fourth. In hospital practice patients often get up on the ninth day, and go home on the thirteenth or fourteenth; but this is usually a matter of necessity rather than of expediency; and when circumstances allow of it, a patient should be directed to lead the quiet life of a semi-invalid for six weeks after delivery, that is, till involution is anatomically complete.

Pathological Section

I. PUERPERAL INFECTION

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Definition. — A wound infection due to the action of micro-organisms, and originating in the generative organs of parturient women. It is not exactly synonymous with the terms puerperal septicæmia or puerperal fever, because it includes not only systemic infections, but also local inflammations.

Etiology.—In considering the causation of this condition we may regard it from three aspects:—(1) The micro-organisms which produce it, or the seeds of the disease; (2) the tissues involved, or the ground upon which the seeds are sown; (3) the ways in which the infective matter is brought to the patients, or the sowers of the seed.

The first of these has been fully dealt with under Micro-Organisms (vol. vi. p. 388), and it need here only be stated that in by far the greater number of cases streptococci are the cause both of the local affections and the general systemic disturbances; out of 91 cases examined by Czerniewski, Widal, and Bumm, they were present in 85, or 94 per cent. The importance of other germs, either alone or in combination, in the production of puerperal infection has not been certainly determined, but there is little room to doubt that such infections have been due, in a certain number of cases, to colon bacilli, gonococci, bacillus pyocyaneus, bacillus fœtidus,

the pneumococcus, diphtheria bacillus, and putrefactive saprophytes.

As regards the second, or the ground upon which the seed is sown, the condition of a pregnant or parturient woman is in many ways more favourable to the development of micro-organisms than at other times. She is in a state of plethora, hydræmia, hyperinosis, and leucocythemia. The total quantity of blood is increased, it is more watery, and contains more fibrin and a larger proportion of white cells, and is loaded with effete matter derived from the fœtus and the genital organs during their involution. The blood-vessels and lymphatics, especially in the genital organs and their immediate neighbourhood, are dilated and hypertrophied, and the nervous system is in a peculiarly irritable condition. The skin about the vulva is always infected with micro-organisms, often of a malignant kind, and the vagina also frequently swarms with microbes, some of which are indistinguishable microscopically from the most dangerous of their kind. The vaginal flora has attracted considerable attention. Döderlein, for example, published the results of his investigations in 1892, from which he concluded that vaginal discharges might be classified as normal and pathological. The normal were met with in something over half the cases examined, and were characterised by a consistence of clotted cream, intensely acid reaction, and by containing a pure culture of an organism which he called the vaginal bacillus, and to which he attributed the production of the lactic acid to which the reaction was due. The pathological discharge was of thinner consistence, often puriform in appearance, sometimes containing bubbles of gas feebly acid, neutral, or even alkaline in reaction, and swarming with micro-organisms, some of which were dangerous. The normal discharges he discovered by experiment to be not only innocuous, but even antiseptic, and capable of destroying staphylococci and other germs introduced into the vagina. Kroenig, who repeated Döderlein's experiments, agreed with him as to the antiseptic properties of the normal discharge, but denied that it was due to the vaginal bacillus, and found that the so-called pathological discharges, which did not contain this organism, also possessed similar bactericidal properties. Menge attributed the germicidal powers of the vagina to: (1) the antagonism of the normal microbic flora to pathogenic organisms; (2) the products of the life process of vaginal bacilli; (3) the acidity of the discharge; (4) the germicidal powers of the anatomical elements of the vagina; (5) phagocytosis; and (6) the absence of free oxygen. Walthard found that the vagina often contained pathogenic microbes, but they were not virulent, and behaved as saprophytes, producing no results when inoculated upon healthy tissues, but becoming virulent when in contact with dead tissues or those whose vitality and powers of resistance had been reduced. From all these investigations we may conclude that the vagina contains germs which are harmless under normal conditions, but which may sometimes become dangerous in the presence of dead matter or tissue whose vitality has been reduced by crushing and bruising in prolonged and difficult labours, and may also be productive of harm if carried upwards into the uterus. The cavity of this organ and the tubes are usually aseptic, and the former is protected from invasion by the cervical operculum. This plug of mucus, though not in itself germicidal, furnishes no nutriment for micro-organisms, yet its lower part is often found to contain vaginal bacteria and leucocytes. The middle portion contains leucocytes only, and the upper neither leucocytes nor bacteria. It may therefore be described as consisting of septic, antiseptic, and aseptic zones. We see then that although the system of a pregnant woman is peculiarly favourable to the growth of septic organisms, yet nature has provided sufficient protection against their invasion under ordinary circumstances. During labour also nature is fully competent to avert evil; and the washing out of the vagina by the liquor amnii, and its further cleansing by the descent of the fœtus and its afterbirth, tend in the same direction, whereas vaginal examinations, the introduction of instruments, and douches counteract these safeguards, and are liable to carry septic matter upwards. In concluding our remarks upon the seat of infection, we must not forget that open wounds or unprotected surfaces are always present in women who have recently been delivered; for not only are the vagina and the cervix frequently torn or abraded, but the interior of the uterus, from which the decidua has been separated, and especially the placental site, must be regarded as a wounded surface.

Thirdly, as regards the modes of infection. The micro-organisms which we have seen are often present in the vagina before delivery are a possible source of mischief, but whether they are ever the cause of puerperal infection has not yet been decided. Semmelweis taught that fever could originate in this way, and described it as auto-infection, and Ahlfeld thoroughly supports this view; but others, considering how infrequently women who are delivered without assistance are attacked by serious illness, deny the possibility of auto-infection. But I think the majority of those who have carefully weighed the evidence on both sides consider that such a mode of infection is certainly possible, though exceedingly rare, and that it might be due not only to the vaginal micro-organisms, but to those also which are occasionally present both in the uterus and the tubes.

In the vast majority of cases the infective material has been conveyed to the patient on the hands of her attendants, or on their instru-

ments and clothing, or on materials used for cleansing purposes. The medical attendant may have been contaminated by suppurating or sloughing wounds, erysipelas, diphtheria, or dead and putrid matter, and the nurse may have handled septic or putrid discharges. Such hands would be peculiarly liable to propagate disease; but it must not be forgotten that dangerous material is everywhere, and that under all circumstances our hands are impregnated with germs, often of a most virulent nature. At one time the air was supposed to be the chief source of infection, but the results of antiseptic midwifery showed that this was not the case, and it then came to be regarded as of little importance. But this is equally erroneous, and there can be no doubt that many cases of putrefaction, if not of actual septicæmia, have been due to air vitiated by decomposing matter, and we cannot safely ignore the importance of a pure atmosphere in a lying-in chamber.

Clinical Features

When infectious material has been introduced into the genital canal its further history depends upon whether the micro-organisms are able to penetrate the tissues or not. In the former case they are generally carried by the lymphatics and blood-vessels to distant parts—SEPTIC INFECTION; whereas in the latter they remain upon the surface, causing putrefaction, and the toxins thus formed cause putrid intoxication or SAPRÆMIA.

We may conveniently classify puerperal infections as follows:—

1. *Vulvitis and Vaginitis.*
2. *Endometritis.*
3. *Parametritis and Perimetritis.*
4. *Septicæmia.*
5. *Pyæmia.*
6. *Endocarditis.*
7. *Phlegmasia alba dolens.*

This classification is artificial, and in practice the various conditions occur in every possible variety of combinations, yet for descriptive purposes, and with regard to prognosis and treatment, it will be found useful.

Vulvitis, Vaginitis, and Endometritis may remain as purely local conditions, or they may be the first steps in a systemic infection. The chief points to be observed in determining this vital question are, the aspect of the patient; her subjective condition; whether she can or cannot sleep; and the rate and condition of her pulse. In general terms, it may be said that if a patient looks and feels well she has not septicæmia.

The wounds in the vagina and about the vulva are peculiarly liable to infection, and readily assume the condition known as puerperal ulcers. These are attended by little or no fever, and the patient seldom complains; but ischuria or smarting when passing water is sometimes present. On inspection one labium is generally swollen, and upon examining its inner surface the ulcer is observed; its base covered with a white or greyish yellow slough, its margins red and swollen, and the discharge thin and irritating, causing excoriation of the surface over which it flows.

Endometritis may be either putrid or septic. In the putrid form the process may be general, or limited to certain areas, especially the placental site. The uterine wall rapidly proliferates a barrier of granulation tissue, which protects the deeper structures from invasion, and secures the separation of the diseased tissue. In septic infection this barrier is imperfect, or absent, and the micro-organisms invade the muscular tissue, the uterine wall becoming soft and swollen, and the disease spreads along the lymphatics to the cellular tissue and peritoneum.

Putrid endometritis is recognised by the character of the lochia, which may at first be scanty, but soon become profuse and offensive. The uterus feels large, and may be tender, and the after-pains are often abnormally acute. The attendant fever is seldom severe, the temperature seldom exceeding 102° F., or the pulse 110 beats per minute. The patient does not look dangerously ill, and her sleep is undisturbed.

Parametritis and Perimetritis.—These conditions are usually ushered in by a rigor, or at least a sense of chilliness, on the second or third day after labour. In parametritis the temperature rises rapidly; in perimetritis more gradually, and the pulse follows the rise of temperature, the after-pains are exquisitely painful, and the uterus becomes tender; at first to one side, but subsequently the tenderness extends over the entire hypogastrium. The duration of the fever varies from days to weeks, but from eight to ten days is about the average.

Peritonitis and cellulitis are usually met with in combination, but it is of importance to determine which tissue is the more involved in a given case, and still more so to determine whether the condition is a local one or part of a systemic infection.

In pelvic cellulitis the temperature rises rapidly, often to 104° F.; the pulse, following more slowly, reaches 100 or 110 beats on the following day; the constitutional disturbance is slight. On bimanual examination a swelling is felt to one side of the uterus, at first diffuse, but becoming harder and more defined; the uterus is pushed towards the opposite side of the pelvis, and as the swelling advances it involves and fixes the cervix; it extends outward to the pelvic walls and iliac fossæ, or backward along the folds of Douglas. Sometimes it extends along the anterior aspect of

the broad ligament, to Poupart's ligament, and down the thigh along the femoral vessels. A localised parametritis usually ends in resolution, but suppuration may occur with hectic fever. In pelvic peritonitis the pain is much more acute and the constitutional disturbance greater. The temperature rises gradually, and seldom exceeds 102° F.; the pulse, on the other hand, rises rapidly, often reaching 120 or more beats per minute. The effusion is less distinct, and is usually found behind or in front of the uterus. When Douglas's pouch is distended the vagina is pushed down before it, forming a well-defined dome, over which the mucous membrane can be easily moved by the finger; whereas in cellulitis this membrane is immovably fixed; the swelling may extend upwards to, or above, the fundus, and pushes the rectum to one side. In cellulitis, on the other hand, if posterior to the uterus, the swelling never extends above the line of firm peritoneal attachment; and sometimes completely surrounds the rectum.

It is of the utmost importance to decide whether these conditions are merely local inflammations or part of a general systemic poisoning. There is seldom any difficulty in deciding this question in the case of cellulitis, but it is otherwise in the case of peritonitis, and the most that we can do, for some days at least, is to hope that a localised peritonitis may remain so, and therefore the extension of tenderness to the upper part of the abdomen is an ominous symptom. On the other hand, a localised peritonitis is generally more acutely painful, and therefore intense hypogastric pain is relatively a good sign in these cases.

Septicæmia

A general infection always commences in one of the local conditions already described, most frequently as an endometritis, but the organisms invade the tissues, and are carried into the system by the lymphatics and blood-vessels, generally by the former alone. There is usually an incubation period of about thirty-six hours before the fever sets in; but since the infection may occur before, during, or after labour, the time when symptoms occur is also variable, but they do so most frequently on the third day after delivery. The fever is ushered in by a rigor or sense of chill; the temperature rises rapidly and remains high, with but slight remissions, until shortly before death. The pulse rises rapidly, generally reaching 140 beats on the second day, and continues to increase until it becomes uncountable. The patient looks and feels extremely ill; her aspect is indeed almost pathognomonic of the disease. The face assumes a nervous, anxious expression, with pallor of the skin, which often assumes an icteric hue; the features become pinched, with hollow cheeks and sunken eyes. She is at first fretful and inclined to tears, with a dread of impending death; she cannot sleep day or night in spite of opiates. The lochia are scanty or suppressed, and in some cases putrid; the vulvar and vaginal wounds are covered with diphtheritic membranes. Peritonitis sets in early and soon becomes general, so that the abdomen becomes greatly distended and tender to the touch. As the disease advances a condition of euphorea sets in, and the patient may feel and say that she is well, though evidently most seriously ill. She often becomes drowsy or somnolent (septic somnolence), though when the disease involves the stomach she is constantly distressed by incessant vomiting, which continues without intermission to the end; the vomit at first consists of the stomach contents, but soon changes to green, brown (coffee ground), or black. The respiration becomes rapid and superficial. The surface is covered with a cold sweat, and the patient dies in collapse usually towards the end of the first or the beginning of the second week.

In the most acute forms of septicæmia the symptoms commence during or soon after labour, and death follows in a few hours. So rapid are some of these cases that there may be no rise of temperature and but few post-mortem appearances.

Diagnosis is seldom difficult. The aspect of the patient, sleeplessness, her small and rapid pulse, and general peritonitis are sufficiently characteristic; it has, however, often been mistaken for typhus or enteric fever, scarlatina, diphtheria, and acute tuberculosis.

Prognosis is generally bad, and the sooner the symptoms occur and the more rapid the extension of the peritonitis the worse it is.

Post-mortem Appearances. — The interior of the uterus and the vaginal and vulvar wounds are covered with sloughs; the uterine wall is swollen and œdematous, sometimes gangrenous. The lymphatics are plugged with thrombi, and on section look greyish-yellow, resembling minute abscesses. Cellulitis is almost always present, the tissue is œdematous, seldom infiltrated with pus, and microscopically shows a small-celled infiltration. This cellulitis is one of the most important lesions in the disease, as peritonitis is the result of the underlying cellulitis, and the general infection spreads along the cellular tissue with amazing rapidity, following the course of the lymph current, first along the ovarian and uterine vessels to the iliac fossæ, upwards to the diaphragm, and into the posterior mediastinum, causing pleuritis and endocarditis, or downwards under Poupart's ligament, giving rise to a phlegmasia alba dolens. The peritoneum is of a dark red colour, especially where it covers the intestines, and its cavity contains a thin, horribly fœtid or purulent fluid. Most of the important organs of the body are affected—the heart, pleura, kidneys,

spleen, and liver. The skin is often icteric or covered with petechia or an erysipelatous blush.

Pyæmia

Metrophlebitis would be a more correct term, because the inflammation spreads from the uterus to the veins, a phlebothrombosis being the essential characteristic, the formation of pus an accidental complication. The streptococci either invade the thrombi already existing in the mouths of the veins, or they first grow along the walls (periphlebitis) and then into them, causing inflammation of the intima, and thereby clotting and thrombosis. The thrombi thus infected break down and are carried as minute septic emboli through the vena cava to the heart and pulmonary system, causing infarctions. In pulmonary abscesses the micrococci themselves may pass through the pulmonary capillaries to the left side of the heart, and from thence be distributed throughout the body, causing abscesses in various parts. The process of thrombosis may extend widely, without the detachment of emboli, involving the pelvic, iliac, and femoral veins. Pyæmia commences as a rule much later in childbed than septicæmia. It has been observed to occur more frequently in cases in which there has been some anomaly in the third stage, either premature expulsion of the placenta by Crédé's method, or its retention and manual removal; also after placenta prævia. It is especially liable to supervene upon a septic endometritis or sloughing carcinoma. The endometritis may have been overlooked, and the practitioner be assured that the patient has been quite well, until a violent rigor at once attracts attention. The patient looks very ill, with pinched features, chattering teeth, and shaking limbs. The temperature rises at once to 104°-105° F. or more; sweating sets in, followed by rapid defervescence to normal or below it; the patient feels relieved and looks well, and unless the condition is complicated by septicæmia (septico-pyæmia) there are none of the alarming symptoms of that condition present. The practitioner who arrives after the alarming symptoms have subsided should not be betrayed into giving too favourable an opinion upon finding the pulse and temperature normal and the condition of the patient good. A single rigor does not, of course, establish the diagnosis of pyæmia, and the disease, when present, may end in recovery after two or three rigors and without any localisation of pus; but usually the rigors return after an interval varying from hours to days, and abscesses appear in different parts of the body. They occur most frequently in the lungs, attended by pleuritic pains, with bloody or purulent expectoration; in the kidneys, causing lumbar pain, accompanied by the presence of albumin or blood in the urine. The spleen is generally enlarged. An abscess may form in the liver. The hepatitis may cause jaundice, but the yellow colour of the skin so frequently observed in these cases is most commonly due to the breaking down of the red blood cells. Not infrequently the joints are affected and become completely disorganised, or the sheaths of the tendons, especially those of the forearm, suppurate. Subcutaneous abscesses occur much more frequently in puerperal than in surgical pyæmia; they form most often in the gluteal region, and as they are seldom associated with abscesses in the deeper organs their occurrence is a favourable sign. The parotid gland, endocardium, and eye are less frequently attacked.

Course and Duration.—Sometimes the disease ends in recovery in a week or two, in others fatally, and the 16th to the 22nd day after delivery is the commonest term in such cases; in others rigors continue for a month or more, but such protracted cases generally recover.

Prognosis is always uncertain; about half the cases recover. The danger depends chiefly upon the importance of the organs affected, and those patients who are affected with subcutaneous abscesses recover as a rule.

Ulcerative Endocarditis

is a form of septicæmia, but as it is sometimes a primary localisation and presents a peculiar train of symptoms it requires separate consideration. It commences with fever, generally ushered in by a rigor; the temperature rises rapidly, and reaches its maximum on the second or third day; it remains high throughout, with moderate remissions, and attended by repeated rigors. The pulse varies from 140 to 160, and is weak and dicrotous. The nervous symptoms are especially prominent: sleeplessness, headache, giddiness, delirium, apathy, and great prostration. There is delirium usually of a low muttering form, though occasionally of a wildly maniacal character, and the patient sinks into stupor and coma. The neck is retracted, with convulsive twitchings, strabismus, grinding of the teeth, and paralysis; a common symptom is persistent diarrhœa. The ophthalmoscope reveals retinal hæmorrhages. Cardiac murmurs are usually absent, but even if detected do not assist the diagnosis, since such murmurs are common in lying-in women; it depends upon the general and especially upon the nervous symptoms, the repeated rigors, and the retinal hæmorrhages. Ulcerative endocarditis differs from ordinary septicæmia in the absence of peritonitis, the early cerebral symptoms, and the longer duration of the disease; from pyæmia in the continued fever without intermissions and the absence of local pus formations. It resembles enteric fever in the constant diarrhœa and splenic enlargement, but differs from it in the course of the fever, the character of the stools, and the negative result of Vidal's test.

Pathological Anatomy.—The left heart is most affected; there are deposits on the mitral and occasionally upon the aortic valves. The myocardium is full of small abscesses, and the endocardium is infected with colonies of bacteria; the endocardium and valves are frequently ulcerated. A number of minute abscesses are found in the kidneys, and there is often suppurative meningitis and abscesses in the brain.

The Prognosis is absolutely bad.

Phlegmasia alba dolens (white leg, or milk leg).—There are two distinct varieties of this condition. In one a cellulitis extends down the thigh, in the other there is thrombosis of the veins with consequent œdema; but many authorities limit the term phlegmasia alba dolens to the cellulitic variety.

Symptoms.—Usually during the second week the patient complains of pain in the affected leg followed by swelling. In the phlebothrombotic form the pain and tenderness are confined to the course of the veins, and the swelling commences in the foot and spreads upwards; in the cellulitic variety the swelling commences in the thigh and extends downwards. The condition is attended in all septic cases by fever and constitutional disturbance, foul tongue, loss of appetite, vomiting, and restlessness.

Prognosis is fairly good in most cases; a compensatory circulation is established, but this is sometimes imperfect, so that the limb remains permanently enlarged. In some cases abscess forms and may lead to pyæmia; or a portion of clot may be displaced, carried to the heart, and obstruct the pulmonary artery.

Prophylaxis and Treatment.—The former of these is by far the more important. Authorities are generally agreed as to its main features and differ only on minor details, whereas in treatment there is hardly a point upon which all authorities agree. The years 1870 and 1880 mark two important eras in the prophylaxis of puerperal infection. In the former year the antiseptic method was generally employed. The patient was regarded as more or less an infected individual, and efforts were made to purify her by douching and various other antiseptic measures. But about 1880 this view was changed, and it became generally recognised that, in the vast majority of cases at least, there was no infective matter to be removed, and that all our efforts should be directed towards preventing its entrance; this is now known as the aseptic method.

There is no doubt that septic infection is most frequently carried to the patient by her attendants, and especially by their hands. This was taught by Semmelweis and Sir James Simpson; and Leopold has clearly proved that patients who have delivered themselves without any vaginal interference, make better recoveries than those who have been examined, even with the most scrupulous antiseptic precautions. We should therefore (1) confine local interference within the narrowest bounds; (2) avoid as far as possible septic contamination; (3) spare no pains to render our hands as innoxious as possible. The attendants should be most careful of their hands; and should a practitioner be called upon to attend patients with infectious diseases or septic and putrid discharges, he should be careful, as far as possible, to avoid contamination; and much may be done in this way by using forceps or rubber film gloves. The nurse who has to wash away putrid lochia or foul discharges should be instructed how to do so without soiling her fingers.

Asepsis is, however, best maintained by substituting, as far as possible, external for internal manipulations. In 1888 Crédé wrote as follows: "Even the simplest manipulations may cause infection. It should therefore be laid down and taught as a fundamental principle, that internal examination of parturient women should be altogether avoided, or restricted within the narrowest possible limits; it can be very well replaced by external examination. To instruct their pupils as thoroughly as possible in this method is the present and future duty of teaching institutions." Internal examinations are, however, occasionally necessary, but the practitioner should remember that such examinations are never altogether devoid of risk, and before resorting to them he should be fully persuaded in his own mind as to the necessity, and he should spare no pains to render his hands as aseptic as possible. This he does in two ways: firstly, by mechanically cleansing the skin with soap, hot water, and a nail-brush; and, secondly, by using some chemical substance to destroy, or at least limit the procreative powers of the micro-organisms in the skin. Of the latter agents alcohol is probably the best, and next to it bichloride or biniodide of mercury. Carbolic acid, chinosol, creolin, and lysol are much employed, but are inferior to the former in bactericidal power. An antiseptic for use in ordinary midwifery practice must not only be efficient, it must also be portable, easily prepared, and not too costly; and all these requirements are fulfilled by corrosive sublimate, which is easily carried in tabloid form. Besides their hands, the attendants should observe the strictest cleanliness in their persons and clothing. With regard to the former, not only are the ordinary rules of cleanliness to be observed, but an individual who is suffering from a purulent nasal, oral, or other discharge, or carious teeth, should not attend women in their confinements. As regards clothing, most nurses now wear clean dresses and aprons fresh from the laundry. But it is not easy for a busy practitioner to provide garments of similar materials; he can, however, remove his coat, turn up his shirt-sleeves, and pin a clean towel in front so as to act as an apron.

The lying-in chamber should be clean and well ventilated, and should not be in close proximity to a water-closet or drain-pipe. The nurse should see that everything likely to be required has been provided, and that they are absolutely clean; to this end she should scrub thoroughly all basins, jugs, bed-pans, and other utensils, and she should provide an ample supply of pure water by boiling it for at least half an hour and preserving it in clean jugs, covered with towels. Water is a generally acknowledged source of danger, and the addition of antiseptics in the amount used for uterine douching is not sufficient to sterilise it; though they are better than nothing in an emergency.

The patient herself should adopt the same precautions, as regards cleanliness, as her attendants, especially frequent warm baths during pregnancy; but during labour they are not advisable, because the water which contains the dirt removed from the surface usually enters the vagina, and cases of fever, apparently due to this cause, have been reported. The external genitals should, however, always be most carefully cleansed and disinfected; formerly vaginal douches were used as a routine, and some obstetricians still employ and recommend them, especially French authorities. At the recent International Congress in Paris, Doleris expressed himself strongly in their favour, but the majority of those who have carefully studied the question have abandoned them as more frequently a cause of harm than good (Runge, Leopold, Krönig, Menge). The argument in their favour is, that infective matter is occasionally found in the genital canal. Against this, however, it is urged that such matter very rarely does harm, that the douche is inefficient for its removal, and harmful because it inhibits the normal bactericidal powers of the vagina, washes away the mucus, and so renders labour more difficult and the vagina more liable to injury, and it may carry infective matter into the cervix and uterus. The strongest argument is, however, the fact that the experience of the vast majority of observers has shown that patients do much better who have not been douched. The management of labour, and especially of the third stage, is of the utmost importance, since the retention of portions of the placenta and membranes is a fruitful source of putrefaction, with its consequent dangers, sapræmic intoxication and pyæmia. (*See* "Labour," vol. v.)

Treatment.—Treatment is both local and general; so long as the disease has not passed beyond the uterus, local treatment is of the utmost importance. Puerperal ulcers were formerly treated very actively—cauterised with chloride of zinc or strong perchloride of iron; but this is unnecessary in ordinary local infections, and useless in systemic poisoning: they are best treated by simple cleanliness, and dusting with iodoform, boric acid, or salicylic acid and starch. When the lochia are offensive a vaginal douche is employed, and if the fever continues the uterus should be washed out. This is a measure not unattended with danger, and requires certain precautions. The instrument employed should be a douche, either a can or Hegar's syphon douche, which can be carried in the obstetrician's bag, but never a Higginson's syringe. The instruments should be absolutely clean, and the water sterilised by boiling for half an hour, and the antiseptic employed should not be a strong poison like corrosive sublimate. Creolin, lysol, or chinosol are perhaps the most suitable; half an ounce of creolin in a gallon of water is an admirable antiseptic solution. The patient should be placed upon her back, a position for many reasons to be preferred to the ordinary lateral obstetric position, because there is less danger of the fluid passing into the Fallopian tubes; the uterus is situated lower, and there is less risk of the tube being contaminated during its passage through the vagina; and also because the operator can see the patient's face, and should the fluid enter the veins he can at once discontinue the process on the first symptom of collapse. The vulva and surrounding parts are first thoroughly cleansed, then the vagina is douched out, a clean glass nozzle being employed, and, in the absence of wounds or ulcers, the surface gently rubbed with two fingers. After this the hands are again disinfected, and a long glass tube, or large Bozeman's catheter, is carefully guided along two fingers, which protect it from touching the vaginal walls, into the uterus, which is then thoroughly douched out. The advantages of uterine douching, though generally acknowledged, are not, however, universally admitted, and Schrader condemns it under all circumstances. The cases in which it is followed by improvement are, such authorities contend, without exception cases of putrefaction, which usually recover without local treatment, whilst the douche might be the means of conveying septic matter from without, or spreading the germs already present, and, by causing uterine contractions, displace the thrombi in the lymphatics and so favour systemic poisoning. Many other accidents have followed its use, such as reopening of wounds already closed, which offer fresh channels for infection; displacement of thrombi in the veins, permitting the entrance of fluid or air; and even perforation of the uterine wall, followed by a fatal peritonitis. But since, on the other hand, simple sapræmic intoxication may prove fatal, the majority of obstetricians employ intra-uterine douches in all cases where the disease is still limited to the uterus and vagina; but they are agreed as to the absolute necessity for thorough asepsis and most gentle manipulation; and also that they should never be employed after peritonitis or parametritis has set in. Should there be any suspicion of retained membranes or por-

tions of placenta, the uterus should be thoroughly explored with the finger, and these removed. Some authorities prefer mopping out the uterus with absorbent cotton-wool to douching, but to my mind the latter is preferable, but in some cases where the putrefaction continues after douching I have seen good results follow a thorough packing of the uterus with iodoform gauze. The gauze is removed after twelve hours, and, if considered advisable, a fresh packing inserted. A more thorough evacuation of the uterus can be effected by the curette, and brilliant results following this treatment have been reported by E. v. Brawn, Borel, Weis, Charrier, Pinard, Pozzi, and others; but it is a method even more open to criticism than the use of the douche, because the puerperal uterus is difficult to curette thoroughly, and if any putrid matter be left it would infect the fresh raw surface; but chiefly because it is liable to injure or remove the barrier of protective cell proliferation and so provide an open door for systemic invasion. Its employment has for these reasons been strongly opposed by Olshausen, Fritsch, Fehling, and others. Whether it should ever be employed is therefore still an open question, but personally I am in favour of its employment, at least in cases of incomplete abortion.

The most radical of all local methods is the extirpation of the diseased uterus. This has been done with a fair measure of success, and is advisable in cases where an adherent and putrid placenta cannot be safely removed in any other way; also where fever depends upon sloughing myomata; but it may also be indicated in cases in which the uterine tissues have been invaded, but the disease has not extended beyond that organ. It is, however, an exceedingly difficult problem to determine whether this is so in a given case, and to avoid unnecessary mutilation on the one hand, and hopeless interference on the other.

Parametritis and Peritonitis.—In the commencement of pelvic cellulitis an ice-bag should be laid upon the hypogastrium, which is afterwards replaced by cold or warm compresses. The patient should be most carefully protected from chill, and should not be allowed to leave her bed for some days after all symptoms of fever have subsided. Peritonitis is treated in a similar manner, but the ice-bag can seldom be borne, and opium is generally required to relieve pain. The practice which has proved successful in threatened peritonitis after operations, namely, to avoid opium and force the bowels to act, is dangerous in puerperal cases, because the movement of the bowels is antagonistic to localisation and incapsulation of the disease, upon which a favourable issue depends.

Septicæmia.—So long as the disease is confined to the genital canal the local measures already described should be adopted, but as soon as it has passed beyond and invaded the system they are absolutely useless. We are not as yet in possession of any antiseptic which can destroy the micro-organisms in the tissues! Quinine, mercury, salicylic, carbolic, and boric acids, as well as the benzoate of soda, have all been tried without benefit, and most of them do harm by disordering the stomach and intestines, and thus inducing loss of appetite and diarrhœa. Most antipyretics are too depressing, and mask the symptoms without any compensating benefit. Some advise purgatives, but they are better avoided, and on the other hand astringents should not be too readily administered to check looseness, which in moderation is a useful eliminative. Our chief object in treatment should therefore be to assist the organism in its contest with the microbes. This we can do by a free exhibition of alcohol, which acts beneficially in various ways: firstly, it is a powerful antiseptic and antipyretic, it strengthens the heart's action, and by checking oxidation reduces destruction of the tissues. It is best administered in the form of brandy, or whisky, port wine, and champagne. The quantities should be large; twelve ounces or more of brandy in twenty-four hours is generally required. Food should be administered frequently in easily assimilable forms; eggs, milk, and beef tea are the most suitable. The employment of cool baths benefits the patient in many ways, by cooling the surface and improving the appetite; where its employment is contra-indicated, a wet pack, or sponging with water and vinegar, may be substituted. These measures are especially beneficial when the skin is dry and hot, the patient restless or delirious, and especially in cases of septic somnolence. The patients, as a rule, feel better after the bath, their circulation and respiration are improved, the skin becomes soft and moist, and they take food more readily. Baths are contra-indicated in peritonitis and metrophlebitis, because the movement of the patient might, in the former case, convert a local into a general peritonitis, and in the latter because of the danger of starting emboli.

Antistreptococcic serum has not fulfilled the high anticipations which attended its introduction, and the majority of authorities have reported unfavourably upon it.

The treatment which appears to me to have met with most success in this disease was formulated by Runge in 1866, and consists in the free use of alcohol, avoidance of internal antipyretics, cool bathing, and nourishing diet.

II. AFFECTIONS OF BREASTS AND NIPPLES

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1. Nipples

(*a*) *Sore Nipples.*—Under this term several diseased conditions of the nipple arising during lactation are included, which are of great clinical importance owing to the relation they bear to mastitis and mammary abscess.

The commonest condition met with is a simple abrasion of the nipple, the result of a loss of epithelium. The first stage of abrasion is a simple erythema. The epithelium is next denuded at several points, leaving the subjacent papillæ unprotected, and if this condition is allowed to go on a fissure of the nipple results.

The denuded areas which follow on a simple erythema are usually at the apex of the nipple, and next most frequently at the sides and base. Basal fissures may extend so deeply, from repeated attempts at nursing, that the nipple may be entirely severed, or left attached merely by a few lactiferous ducts.

The *predisposing causes* of sore nipples are:—shortness of the nipple, undue thinness of the epithelium, and prominent papillæ with deep intervening sulci, which tend to imprison shed epithelium and drops of milk; these combining to form a crust, the non-removal of which favours atrophy of the underlying epithelium. Neglect and want of cleanliness during pregnancy, and pressure of tight corsets, which may prevent the development of the nipple, are important etiological factors.

The *direct cause* is injury set up by the gums and tongue of the infant during suckling. This is most apt to occur if suckling be delayed until the breasts are engorged, if the entire nipple be not taken into the child's mouth, or if the nipple is not properly developed, or is depressed.

The unprotected papillæ bathed in milk and blood form an excellent nidus for the culture and growth of micro-organisms. Bacterial infection may result from the fingers of the nurse or mother, from the child's mouth, or from soiled clothes, and thus what was originally a simple irritation is converted into a septic wound. Bacilli and cocci, including the *staphylococcus aureus* and *albus*, have been found by Bumm in fissures of the nipple. The *oidium albicans* in cases of thrush, and micro-organisms, may be transmitted directly from the mouth of the child, and it is possible that some forms of stomatitis in the child may be caused by micro-organisms present in the fissured nipple.

Fissured nipples are more common in primiparæ, and in women with delicate skins, especially blondes. Abnormal states of the mother's milk may also predispose to fissure of the nipple, or aggravate an already existing fissure.

Symptoms usually begin from the third to the fifth day. Excruciating pain is complained of when the child is put to the breast. The temperature may rise to 104° F., either as a result of septic absorption or from the deep impression made on the nervous system by the intense pain. The nervous apprehension of the pain may even cause convulsions to supervene, and may seriously interfere with the patient's general health.

Treatment.—Prophylactic treatment should begin during the last two or three months of pregnancy, when special attention should be directed to the avoidance of pressure by corsets. The nipples should be washed night and morning with soap and cold water, and then bathed with some astringent or spirituous fluid, such as solution of alum, tincture of quinine or arnica, brandy, whisky, or eau-de-Cologne. Small depressed nipples should be gently pulled out daily with the finger and thumb, which serves to stimulate and bring into action the muscular fibres. Inunction with lanoline or simple ointment facilitates the pulling out of the nipple. Care should be taken not to over-manipulate the nipple late in pregnancy, as uterine contractions may thus be set up.

Depressed or invaginated nipples may be pulled out by means of the breast-pump, or oral suction by another healthy woman, or by the mouth of a bottle just emptied of hot water. Strips of adhesive plaster may then be carefully and judiciously applied to retain the nipple in its normal position (Henry Davies). In severe cases, Kehrer's operation of mammillaplasty may be performed.

Attention should be paid to the cleanliness of the nipple and of the child's mouth from the beginning of lactation. The child's mouth should be washed at least once daily with a saturated solution of boracic acid. Before and after each act of suckling the breasts should be washed with boric solution, or 1 in 2000 corr. sublim., and thoroughly dried. If the nipples are carefully dried the child will only absorb an infinitesimal quantity of mercury.[1] If any erythema is present a

[1] The child should not be put to the breast too frequently, and each nursing should be limited to fifteen minutes at longest.

protective ointment should be applied, such as ung. bismuthi alone, or mixed with an equal quantity of boracic acid ointment. Compound tincture of benzoin or hazeline are also useful applications, which may be used alternately with the ointments. If there be erosion, nothing acts so well as ichthyol ointment, 15 to 20 per cent with lanoline, or iodoform ointment, gr. x. to ℥j. Dusting with powdered tannic acid, or painting with a solution of silver nitrate, 2 per cent, may often be found useful.

For a deep fissure a fine pointed stick of silver nitrate should be applied. A solution of chloral hydrate (4 per cent) may be tried, preceded by the application of a 5 per cent solution of cocain.[1] Corrosive sublimate solution, 1 in 2000, should also be used as a wash.

To give time for the healing process to occur, a nipple shield should be used if it can be borne, and if it does not tend to aggravate the fissure. If the shield cannot be borne, the breasts may be relieved artificially by massage, which may be continued for four days, nursing being again attempted at the end of that period. If a nipple shield is used it must be kept scrupulously clean, and when not in use should be kept in boracic acid solution.

A slight fissure will usually heal up in about twenty-four hours with proper treatment, but in severe cases it may take longer. It is rarely necessary for the mother to give up nursing entirely, unless a mammary abscess is imminent, or unless the pain and distress are so intolerable that the patient cannot persevere.

(*b*) *Deep Inflammation and Abscess.*—Inflammation of the deeper part of the nipples is rare. It may attack the lactiferous ducts or the connective tissue of the nipple, and may end in resolution or in the formation of a small abscess. If pus forms in the ducts, it becomes evacuated through the normal apertures, and is apt to be swallowed by the child. If the connective tissue is involved a painful abscess is apt to form, which may either burst spontaneously or require incision, after which it usually heals rapidly with antiseptic treatment. Lactation should be stopped for a few days. In using the bistoury the greatest care must be taken not to divide a milk duct transversely.

(*c*) *Eczema*, *scabies*, *herpes zoster*, *diphtheria*, *thrush*, and *syphilitic ulcers* may affect the nipple. Eczema will be considered under affections of the areola. Syphilitic ulcers are to be treated on the ordinary principles of general and local treatment. If the child presents no symptoms of syphilis it should be weaned. These ulcers may be difficult to recognise as syphilitic; in such cases the enlargement of the axillary glands, sore throat, and a faint roseola on the skin should be carefully looked for.

(*d*) *Anomalies and Defects in Conformation.*—The nipple may be absent congenitally, or from the result of injury or suppuration in infancy. The nipples may be small, ill-developed, or depressed, and there may be supernumerary nipples. The greatest variety exists in their development in different women. Some are bilobed or have an exaggerated foliaceous arrangement of the papillæ with deep clefts and divisions. These latter are more apt to excoriate and crack during lactation, for the reasons already given; and depression or invagination of the nipples is probably one of the most frequent factors in the etiology of mammary troubles during lactation. These defects should be remedied as far as possible by treatment during pregnancy, as already described.

2. Affections of the Areola

(*a*) *Eczema* of the areola and nipple is more frequent in blonde women with delicate skins. It is often difficult to cure, and is apt to be obstinate under treatment, possibly from the presence of a staphylococcus in the milk, which is constantly reinfecting the areola (Stumpf). The predisposing causes are the moist and sodden condition of the areola, the result of nursing, the friction of clothes, and the entrance of micro-organisms into small excoriations or fissures. It may also arise from a discharging sinus, causing skin infection in a neglected case, combined with the results of poulticing in such cases. It is characterised by much itching and burning, by a moist discharge which stiffens linen and forms yellowish crusts. It is apt to spread to the adjacent skin, and if neglected may lead to fissure and mastitis.

Treatment.—If possible the condition should be prevented from becoming chronic. To this end the parts should be kept as dry as possible, and the application of the powdered oleate of zinc produces, as a rule, gratifying results. In bad cases lactation should be stopped, or a gutta-percha nipple shield may be tried. The crusts should be removed by bathing them with either a weak solution of bicarbonate of soda, to which oatmeal flour is freely added, or warm rice-water, or a boracic starch poultice may be applied. After the crusts are removed in this way various applications may be tried. When there is much hyperæmia mild soothing remedies should be used, and more stimulating treatment later. Ointments of zinc, salicylic acid, carbolic acid, *liquor carbonis detergens*, or ichthyol, and diluted citron (Velpeau) or sulphur ointments. More stimulating remedies, as resorcin, nitrate of silver, and corrosive sublimate, may be used later. When acute symptoms have subsided an ointment of nitrate of lead

[1] Films of Unna's glycerine jelly form good protective applications, with which may be incorporated ichthyol, carbolic acid, resorcin, boric acid, lead, zinc, tannin, etc.

(gr. x.-xv. to ℥j.) is very useful (Fordyce Barker).

(*b*) *Cellulitis, adenitis*, and *abscess of the areola* are simply an extension of the similar condition from the nipple (already considered). This is rarer than suppuration within the breast itself. The condition is sometimes called supra-mammary abscess (abscès de l'aréole). When suppuration occurs it is better not to incise till a distinctly pointed swelling is evident, in case of injuring a milk duct.

Proper attention to the nipples to a great extent prevents this condition, and resolution generally occurs if the exciting cause, the irritation of suckling, be removed.

Small boils or pustules may develop in the sebaceous follicles of the areola from suppuration.

(*c*) *Anomalies of Areola.*—The writer has met one instance where suckling was rendered impossible by the escape of milk from Montgomery's tubercles while the child was at the breast.

3. Affections of the Mamma

(*a*) *Mastitis*, or inflammation of the breast, commonly called a "weed," may affect both breasts simultaneously, but more frequently affects one (usually the right). It is more common in primiparæ. Blondes and lymphatic subjects are most subject to it, and it is rarely met with after the fourth pregnancy.

Since more attention has been paid in recent years to prophylaxis, its frequency has fallen from about 6 per cent to 3 per cent in confinements in private practice, and at the present time a case is almost unique in a lying-in hospital.

There are four periods during the puerperium when mastitis is most likely to occur :—

(1) In the first month—the stage of mutual adaptation.

(2) When there is sudden arrest of suckling, causing liability to milk stasis.

(3) At the period of appearance of the first teeth, when the nipple is exposed to injury.

(4) At the period of weaning, when engorgement may occur from irregular emptying of the breasts, or when the child, failing to get nourishment, shows its dissatisfaction by tugging at the nipple.

Varieties of Mastitis.—(1) Parenchymatous or glandular (the most frequent form).

(2) Subcutaneous.

(3) Postmammary or subglandular.

These varieties may coexist, and frequently a mastitis begins as a parenchymatous form, and then approaches the skin surface of the gland.

Causes.—The puerperal breast, being an organ frequently engorged with blood, and containing an animal fluid, forms consequently a convenient nidus for the proliferation of organisms. The dependent position of the glands further favours congestion, and owing to their situation they are liable to injuries. It is now abundantly proved that mastitis is invariably caused by the action of organisms. Mere stasis of the milk will not produce inflammation, unless the milk contains abundance of bacteria. Kehrer has sealed up the nipple with collodion, and no inflammation ensued from the resulting engorgement and stasis. Stasis is a frequent precursor of mastitis, but never in itself a cause.

Organisms can gain access to the breast through its numerous ducts when there are any abrasions on the nipple or areola, and as such breaches of surface are frequent in lactating women, the entrance of pyogenic organisms readily takes place. Nowhere are the usual organisms of the skin found in greater numbers than in the axilla and on the mammary areola, and micro-organisms exist in special abundance in the moist fossa at the base of the nipple. These organisms are mostly pyogenic, and thus it is not to be wondered at that the least abrasion of the nipple becomes a septic wound. Organisms may also be conveyed to the nipple by the child's mouth, the hands of the mother, a dirty nipple shield, etc. Sometimes the lesions on the nipple through which absorption has taken place are so small as to require a lens to distinguish them.

Micro-organisms have the special power of coagulating caseinogen on gaining access to the milk. Escherich believes that, in puerperal septic conditions, microbes, which have entered the blood from lesions in the genital canals, may find their way into the milk and set up mastitis. Hence the importance of keeping the genital tract aseptic.

Congestions and Milk Engorgements.—Many of these conditions are purely physiological, as when the milk first appears, and are accompanied sometimes by pain and slight rise of temperature. Milk engorgement may occur when suckling is suddenly interrupted, as by the death of the child, or when nursing has become painful from tender nipples. The breast then swells, becomes hot, painful, and congested, while localised areas of induration can be felt, which are loaded milk ducts. The superficial veins become large and prominent. This condition, especially if associated with neglected sore nipples, is almost certain to go on to mastitis and abscess formation, owing to the easy absorption of organisms.

Various causes may interfere with the milk flow, thus leading to engorgement in the milk ducts of a part, or even the whole, of the breast, *e.g.* hyper-secretion, chill (which may occur quite as readily from the patient's night-gown being soaked from an overflow of milk through the night, as by direct exposure), or blocking of a duct by some inspissated secretion.

Treatment of Mammary Engorgement.—In the first place any abrasion or sign of excoriation about the nipples must be rendered aseptic with a mercurial lotion. In mild cases some

evaporating lotion, as eau-de-Cologne or vinegar diluted with water, gives relief. In addition, graduated pressure should be adopted by means of layers of cotton-wool kept firmly applied by bandages, or by an ordinary tightly fitting slip-bodice. If the breasts are pendulous, slinging them in a silk handkerchief is a great comfort if pressure cannot be borne. Suckling on the affected side must be desisted from, and a breast-pump, or preferably careful massage, substituted. When there is great engorgement more vigorous measures are required. Free saline purgation, antiseptic fomentations, and a dry and restricted diet are the first indications. The breast-pump should be used, and the breast massaged from the base towards the nipple with warm olive oil alone, or combined with an equal part of belladonna liniment. Frequently the breasts can be kept empty by means of massage when the application of the breast-pump is painful and unsatisfactory. The breast-pump only withdraws milk from the large ducts near the nipple, so that to empty the breast thoroughly the use of the pump should be combined with massage. If the child is off the breast, belladonna or atropine can be given internally, combined with full doses of iodide of potassium. Sculptor's clay has been recommended as an application in mastitis. It is spread thickly under a bandage over the breast, leaving the nipple free, and is renewed morning and evening.

The temperature may rise to 102° F. or 103° F. in simple engorgement, and thus simulate abscess formation.

(*b*) When symptoms of abscess show themselves, massage and compression must cease, and the abscess is treated on ordinary surgical principles.

(*c*) *Milk Nodes.*—Owing to the constriction of one or more lacteal canals, inspissated milk may collect and remain for an indefinite period, forming swellings in the breast.

Treatment.—Promote their absorption by massage, with the application of a 5 per cent oleate of mercury ointment.

(*d*) *Galactocele* is the result of permanent occlusion of a lactiferous duct. The retained milk forms a cystic tumour which may require surgical interference.

(*e*) *Fistulæ* may be of two kinds—first, the result of an abscess which has not closed; and second, a communication between the skin and a lacteal duct, caused by injury to the duct by a penetrating abscess, or by the surgeon's knife. In either case lactation should be given up.

(*f*) *Anomalies.*—Congenital absence of one or both breasts may occur, or more frequently there may be a small breast which is valueless for lactation. There may be supernumerary mammary glands, which for the most part are functionless.

(*g*) The puerperal mamma may be affected with tubercle, erysipelas, and molluscum contagiosum, the latter derived from the child's face.

4. Conditions interfering with Suckling

See also "Infant Feeding," vol. iv. p. 389.

(*a*) *Artificial Arrest of Lactation.*—Lactation may be arrested at three periods:—(1) Immediately after delivery, before full secretion has been established; (2) during the course of lactation at any time; and (3) at the end of the period of lactation. The secretion can be most easily arrested, and with least disturbance to the patient, at the first or third of these periods.

Means of Arrest.—In the first period this can generally be accomplished by (α) pressure, as already described under the treatment of "mastitis," if possible before engorgement has begun; (β) purgation—early administration of saline purges—and a "dry diet"; (γ) gentle massage; (δ) abstraction of a small quantity of milk by the breast-pump to prevent dangerous engorgement during the first few days; (ε) iodide of potassium (gr. xx. to xxx.) and belladonna internally; locally some preparation of belladonna, or atropine ointment (gr. j. to ℥j.). Belladonna plasters may be applied in such a way as to keep the nipple uncovered. Plasters, however, sometimes cause irritation, and inflammation may go on unrecognised beneath them. A plaster 6″ × 6″ is sufficient for each breast.

In the second period compression, free purgation, and dry diet are the chief means of arrest. After massage and suction during this period the breasts tend rapidly to refill, and therefore such treatment is not so satisfactory in this as in the first stage. If, however, nodular masses form in the breast, indicating insufficient milk drainage, early massage is necessary to prevent the possibility of mastitis. If the massage causes pain, the application of a mixture of equal parts of glycerine and extract of belladonna, over which hot fomentations are applied, gives great relief, and so permits of the subsequent use of massage. Internally iodide of potassium and belladonna should be given.

In the third stage the arrest of the milk flow is simply natural weaning, and is discussed under that heading in the article on "Infant Feeding." Any slight tension of the breast which may occur at this time, when a "nursing" has been omitted, can be reduced by gentle massage with or without a short application of the breast-pump.

(*b*) *Abnormalities of Human Milk.*—These have already been fully discussed in the article on "Infant Feeding." It only remains to be added that healthy mothers' milk does not come from the breast absolutely sterile. The micro-organisms commonly found are *staphylococcus pyogenes albus* and *aureus*, which probably find their way into the milk from without through the milk

ducts. On the other hand, as already stated, organisms may reach the milk through the blood in women suffering from septic diseases. The cocci which may be ordinarily found in milk produce no ill effect, and they seem to be destroyed by the bactericidal action of the intestine in healthy children. Weak children do not possess this action to the same extent, and so the cocci may flourish in the intestine and produce various infantile disorders. If the milk contain more virulent organisms, as in mastitis and puerperal fever, the child may be seriously affected.

Blue milk is due to the presence and development of the *bacillus pyocyaneus*. Red milk may be due to the presence of blood, or may result from the action of the *micrococcus prodigiosus*, which sometimes grows with great rapidity in milk allowed to stand, giving it the appearance of blood.

Green and yellow milks are also organismal in their origin. Bile pigment and pus may pass into the milk. In all such cases weaning is necessary.

Jorisenne has recorded a case of fœtid milk in the mamma, when the milk became fœtid if there was any unduly long interval between the woman's nursings, becoming sweet again when she nursed regularly. The child was sick when the milk was fœtid, but the mother suffered no discomfort except from the smell.

Closely allied to the bacteriology of milk is "Lactation Immunity," which, though of great interest and importance, hardly falls within the scope of this article. A good account of this subject is to be found in the *Science Progress* for May 1894, by Buckmaster.

III. FEVER IN THE PUERPERIUM, ETC.

FEVER IN THE PUERPERIUM DUE TO CAUSES OTHER THAN SEPTIC INFECTION—
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1. FEVER IN THE PUERPERIUM DUE TO CAUSES OTHER THAN SEPTIC INFECTION

A rise of temperature in the puerperal state should always be regarded as evidence of some septic infection unless there is definite proof to the contrary. Low continued fever for a week or ten days without any very severe constitutional disturbance, often attributed to the changes in the mammæ connected with lactation, is much more often due to a subacute inflammation in the pelvis. It is this manifestation of sepsis in the puerperium which is so frequently at the time overlooked, but which is the cause of so much suffering in parous women, and accounts for a large proportion of those chronic cases which fill the cliniques of the gynecologist.

Fever apart from sepsis does, however, occur. It may arise from reflex irritation. The best example of this is the fever which, especially in neurotic patients, sometimes accompanies the engorgement and distension of the breasts about the third or fourth day. It is seen also when the nipples are cracked and fissured, though here it is less easy to eliminate a septic element. The fever, unless mastitis actually sets in, quickly subsides as the breasts become softer when suckling is fairly established, and is relieved by a mild saline purge and gentle massage of the breasts with warm olive oil.

Emotion may be a cause of fever in the puerperal as in the non-puerperal state, and in the weakened condition of the puerperal patient is specially apt to be so. The sudden announcement of some bad news, the death of the baby, the dread of exposure in unmarried girls, worry over the non-arrival of an expected letter, are instances of recorded cases of this so-called emotional fever.

Acute constipation can also undoubtedly give rise to very considerable febrile disturbance in the puerperium. The temperature may be very high, and from its sudden onset the cause may be overlooked. The heavy breath and furred tongue will indicate the treatment, and speedy relief will be got by the administration of two grains of calomel followed in a few hours by a dose of castor oil.

A febrile reaction may follow a sudden exposure to cold. The fever may be ushered in by a rigor; it at any rate follows suddenly on the exposure, so that the history makes the case clear. It is of short duration, and is speedily relieved by the obvious treatment of a hot drink and the comfort of a hot-water bottle.

Any of the acute or chronic febrile diseases may, of course, attack the pregnant woman, and may persist in the puerperal state and form a serious complication. The cause of the rise of temperature in the great majority of these cases will be obvious. In some, however, the original disease is easily overlooked, and the fever may be regarded as septic in its origin. An acute exacerbation of fever, so often observed in phthisical patients during the puerperium, may cause anxiety in this way. It must be remembered also that zymotic disease in its incubation stage, before the fever has developed, undoubtedly predisposes to the onset of labour. Two cases of fever in the puerperium in the writer's experience, at first regarded as septic, proved ultimately to be typhoid, and in a third case, during an epidemic, a smallpox rash which developed on the third day cleared up the diagnosis. All these patients died. It was only

after the correct diagnosis was made that any history of indisposition previous to the labour could be got from the patients or their friends. Influenza attacking a puerperal patient may for a time be difficult to differentiate from a septicæmia.

Fever in the puerperium is fortunately then not always due to sepsis, but, as before stated, great care must be taken before assigning to it any other cause. An early intra-uterine douche, carefully given, in a doubtful case can do no harm, and may do much good in averting a general infection, while, if the operation is followed by a marked reduction in the temperature, it will form a valuable aid to diagnosis.

2. Sudden Death in the Puerperium

Puerperal Venous Thrombosis and Embolism.— During pregnancy the blood contains an excess of fibrin, the amount increasing as gestation advances. This increase of fibrin probably plays an important part in favouring coagulation of the blood in the uterine sinuses after labour is over. It has, however, also a pathological interest, as it predisposes in the puerperium to thrombosis in other parts. The commonest sites are the iliac and femoral veins, but it may occur, according to Playfair and others, primarily in the pulmonary arteries. The separation of such a thrombus and its entrance into the circulation, and its final arrest in some branch of the pulmonary artery, or the formation of a clot in the right side of the heart and its gradual extension along the pulmonary artery, are the causes of the majority of those cases of tragic and sudden death which are met with in puerperal women. The accident rarely occurs before the end of the second week, and, as a rule, there is no warning that anything is wrong. The patient is apparently in perfect health, when suddenly she is seized with intense dyspnœa and pain in the chest. The face in some cases is pale, in others deeply cyanosed; in all it is expressive of great agony. The heart's action, at first exaggerated and irregular, soon becomes feeble and flickering, and the patient dies in a few minutes.

This is the general termination of cases of pulmonary embolism, but in some cases, after a severe attack of dyspnœa, the symptoms slowly abate and recovery takes place. In such a case the thrombus, which is small and blocks only a smaller branch of the artery, is gradually absorbed. In still another class of cases there may be a temporary alleviation of the distressing symptoms, followed later by increasing dyspnœa and cyanosis, and death in a few days. Here there is probably a gradual thrombosis extending from the site of the embolism.

As a rule, death is so sudden that the question of treatment does not arise. In the rare cases not immediately fatal, stimulants should be freely given by the mouth or hypodermically, and mustard placed over the heart; and after the acute distress of the attack has passed the patient must be kept at absolute rest, all attempts at movement being strictly forbidden. Prophylaxis is of greater importance. In the treatment of a case of puerperal thrombosis, as in phlegmasia alba dolens, the affected limb must be swathed in wool, and kept absolutely at rest, it may be for weeks, until the thrombosis has become so firmly attached to the vessel wall that its separation is impossible. Even gentle massage of the affected limb is always dangerous until six weeks from the time the phlegmasia was first noticed.

A much rarer cause of sudden death than that mentioned is the entrance of air into the circulation. The place of entrance is generally the open uterine sinuses at the placental site. The accident has followed intra-uterine manipulations, as in turning, and during the process of intra-uterine and also vaginal douches, where air has been forced in along with the fluid.

The spontaneous entrance of air into the uterine sinuses must be very rare, though such cases have been recorded. The symptoms are somewhat similar to those met with in pulmonary embolism.

Sudden death in the puerperium may arise also from heart disease, from rupture of the heart, from rupture of an aneurysm, or from cerebral hæmorrhage. Many patients die from a sudden acute uterine hæmorrhage, while in others the cause seems to be shock or syncope following a tedious labour.

3. Puerperal Hæmatoma

An interstitial effusion of blood into the tissues of the pelvis, due generally to the rupture of a vein and the formation of a circumscribed tumour, is a rare, but when it does occur may prove a very serious, complication of the puerperium. The situation of the blood tumour varies. In the majority of the cases it is in the vaginal wall near the vulvar orifice, through which the swelling may bulge. It may, however, form in the cervix, in the peri-uterine connective tissue, or under the skin of the perineum, from which it may extend posteriorly into the gluteal region or anteriorly to the abdominal walls. It occurs generally spontaneously, and is then due either to the increased venous pressure which is present during labour, causing rupture of a diseased vessel, or to the direct pressure of the fœtal head in its descent through the pelvis. A varicose condition of the veins would seem likely to predispose to this accident, but this is denied by most authorities. It may be caused artificially by direct injury of a vessel in using forceps. Not infrequently the hæmatoma is noticed during the labour, but more often the pressure of the descending part against the side wall of the pelvis prevents any effusion till labour is over, when it slowly increases in size, and may continue doing so without rupture

for twelve or even twenty-four hours. In rare cases the tumour does not develop until the second or third day. In these cases the rupture of the vessel is due to what has been called a pressure necrosis.

The size of the swelling varies. Generally no larger than the fist, it may be as big as a large cocoa-nut. Pain is generally a prominent symptom, the severity of which depends largely on the size of the tumour. By pressure against the bladder or rectum it may give rise to severe tenesmus and straining. Where the effusion of blood is diffuse and not circumscribed, or where the tumour is very large, there may be symptoms of acute hæmorrhage.

The diagnosis is, as a rule, easy, because in the majority of cases the tumour is visible and can be noticed to slowly increase in size. The swelling has, however, been mistaken for the placenta, for inversion of the uterus, for prolapse of the uterus, and even for the fœtal head.

Prognosis.—(*a*) As a rule the effused blood is slowly absorbed; (*b*) rupture of the tumour may take place and recovery follow; (*c*) the formation of an abscess, the result of septic infection, without rupture, or septic infection after rupture; (*d*) death from hæmorrhage, with or without previous rupture of the tumour.

Treatment. — Should the tumour develop during labour and form an obstruction to delivery, it must be incised and the blood-clot removed. If the bleeding vessel can be secured, the case can be left alone till the labour is over; but if, as is more likely, this is impossible, then forceps should be applied to bring the head down into the vagina, till by its pressure over the site of the tumour the hæmorrhage is controlled. After delivery another attempt may be made to ligature the bleeding vessel. If this cannot be found, then the cavity must be tightly packed with iodoform gauze and an ice-bag applied to the parts externally. There is a great risk of a septic infection, of course, from the decomposing lochia where it has been necessary to incise the tumour, so that the greatest care is necessary to keep the parts sweet by antiseptic douching and bathing of the external genitals.

Should the tumour develop after labour, then treatment should be directed towards controlling the hæmorrhage by pressure and the application of cold. Pressure can be brought to bear over the swelling by the introduction into the vagina of a large Barnes bag; an ice-bag should also be placed over the vulva, and any straining, if possible, prevented. If by these means the hæmorrhage is stopped, as shown by the tumour ceasing to get larger, then absorption of the effused blood will, as a rule, go on slowly. To lessen the risk of abscess formation which may result from septic infection, the vagina must be frequently douched and the external part frequently cleansed with some antiseptic lotion.

If the swelling, once formed, rapidly increases in size, indicating a severe hæmorrhage, then all attempts by pressure and cold may fail to arrest the bleeding, and in order to prevent the sac rupturing it may be necessary to incise the swelling and search for the torn vessel. This will, however, be rarely necessary, and should, if possible, be avoided, because of the risks of septic infection. In some cases where the sac has not been opened, in spite of all care the effused blood will become septic and suppuration supervene. As soon as this is diagnosed a free opening must be made into the most dependent part of the swelling, all blood and pus cleared out, and the cavity, after cleansing, packed with iodoform gauze, which is changed at least once each day.

IV. PUERPERAL INSANITIES

1. Observations on the Nature, Etiology, and Pathology of the Insanities of Pregnancy 376
2. The Clinical Varieties—
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(Note.—Puerperal insanity is the most dramatic and significant variety of the mental diseases associated with child-bearing, but it can only be properly considered as one of a group to which it gives its name.)

1. Nature, Etiology, and Pathology.—Insanity always signifies some dyscrasia—a more or less profound nutritive disturbance in general, some incompatibility in the ingredients which the blood supplies for the tissues' nourishment. Hence follows a loss of balance between waste and repair. That may be what I have called in the general article on "Insanity" (vol. iv. p. 479) a state of "surfeit," or it may approach to a more simple anæmia; but in any case the result is inanition and irritation of the nervous system and of the other tissues. But, besides the general physiological instability, a particular mental instability is also implied by insanity—some failure of balance between the various nervous activities which contribute to mental life, some discrepancy between sensibility and the capacity for effort. No form of insanity illustrates so well as do the insanities of pregnancy these aspects of mental pathology—perversion of nutrition, reflex irritation, and excess of mental effort. There are three recognised varieties of insanity of child-bearing—the pregnant, the puerperal, the lactational—though they are best regarded as a serial development of a protracted process. Each of the three has its characteristic dyscrasia. An account, even approximately exact, of the chemistry of these states is still quite impossible. Very great

importance is now being attached to the factors of auto-intoxication in the etiology of insanity, and quite properly. But we do well, I think, to associate our newer knowledge and impressions with the old conceptions of malnutrition. In particular, I would emphasise the importance in the cases now considered of simple starvation of the cerebral tissues either from some poverty of the blood, for instance, or from defective cardiac action, as well as of the presence of pernicious by-products of metabolism or of defective elimination. We cannot dissociate these factors in etiology. As Dr. Lackie points out (p. 228), many of the changes in pregnancy formerly called physiological are now regarded as due to toxæmia; and we must take the wide view of child-bearing as a process which at once depletes and poisons, exhausts and upsets, the nervous system and every other system. During pregnancy the maternal organism is called upon to bear the burden not only of its own metabolism, but of that also of the fœtus, and we must bear in mind the great frequency, especially in primiparæ, of disordered digestion and impaired excretion, dyspepsia, a poor or perverted appetite, constipation, possibly some uræmia, "hepatic inadequacy," and muscular inertia from insufficiency of outdoor exercise. In the puerperium the maternal economy, suddenly called upon to adjust itself to a quite altered blood-supply on the escape of the fœtus, often finds itself hampered by having to deal with the products of something like septic absorption. And during lactation, just following the strains of the pregnant and the puerperal metabolism, the constitution is drained by a novel, continuous, and expensive secretion. But the nervous factors are also important—the reflex irritations which are inevitable, and the nervous reactions which are not less so, both of which imply nervous differentiations appropriate to the maternal state. We cannot but admit the nervous strain of pregnancy, however well managed, however perfect the metabolism, however aseptic the various tissues—the variety of impulses which beset the brain, from a growing and quickening tumour, partly subconscious, partly impressed upon the attention forcibly or even painfully; the acute impressions of delivery, the sudden pelvic emptiness, the cessation of fœtal impulse, and in its place the painful irritation of a uterus in involution; then another reflex stimulation, usually painful, when the mammæ have to suffer periodic tension and relief.

And again we must attach importance to the mental condition incidental to child-bearing. The emotional state during pregnancy is one of tension. The young mother is usually apprehensive, fearful of some accident to herself or to her child; ready to foresee domestic trouble, or even catastrophe; distressed by her physical incapacity, and apt to feel herself a burden; fanciful and nervous, ready to believe that she is no longer acceptable to her friends, and that she has lost her husband's love. In the puerperal state a great mental reaction occurs. It is usual to have emotional relief; conjugal feeling is forgotten in the maternal, which develops with an intensity that is frequently ferocious, and the new-made mother is actively inclined and restless, itching to be up and about, and as keen to be living an active life as a hen that has hatched a brood. We must note here that this reaction shows itself in insanity, so that, while the type during pregnancy is melancholic or delusional, as it also is during lactation, the insanity of early puerperium is one of acute mania. During lactation, again, the conditions are those of a reaction. The return to active life has proved exhausting, and the patient is weary in body and in spirit; her happiest expectations are not being fulfilled, and her courage is being sapped by the constant drain on her physical resources; very easily the patient slips from the hopefulness and sense of energy of the puerperium to a state of depression and annoyance.

For those who take a broad view of these insanities, and understand the manifold factors to be taken into consideration, I would cite these as very significant facts—that the risk of insanity is increased in cases where morning sickness has been intractable, or where uræmia has been considerable; in those also in which the puerperal processes have gone septic, and in the mothers of illegitimate children. These facts severally illustrate the importance of starvation, of auto-intoxication, of auto-infection, and of worry in the production of insanity at this period. I have dwelt disproportionately upon this aspect of the subject because it is of the utmost importance in treatment. To manage his cases properly from the point of view of preventing insanity, the practitioner must, if he has a pregnant patient who is of a neurotic type, and especially if her heredity or her history makes him afraid lest insanity supervene, he must have regard to conditions which deprive the patient of sufficient nourishment, to those which poison her, whether by non-bacterial or by bacterial products, and to those which add to the already great mental strain of the occasion.

The pathology of these insanities is indeterminate. Of the acute forms—for instance, an acute melancholia of pregnancy or an acute mania of puerperium—the histological appearances would likely be proliferative and degenerative changes in the endothelial elements of the membranes, connective tissue proliferation in the adventitia, and infiltration with leucocytes in the vessel walls, and especially primary degeneration of the cortical nerve-cells, varying with the gravity of the case, both in its degree and in the extent of its distribution. One

would also find in these acute cases after death intense congestion of the cerebral tissue in areas and softening patches. These appearances, however, are common in all acute insanities. The topical pathology is all-important, and we know nothing of it; yet there must be some physical counterpart, for example, of the almost constant change in appetite and affections which characterise these insanities. Also, the chemical pathology is unknown; the products of para-digestion which presumably occur in the dyspepsia of pregnancy, the toxins which are developed in the stomach and bowels, the bacteria and their products which develop either generally or locally in such cases, are not distinctive. It is their presence in the organism, along with the almost constant nervous conditions of these cases, which determine their characteristic results. In short, the insanities of pregnancy are to be regarded as occasioned by malnutrition —by inanition, or by toxæmia, or by both, occurring in nervous subjects at a time when they are already subjected to the strain of nervous developments characteristic of incipient or actual motherhood. As we shall see, these factors can and should be dealt with in treatment, especially in its preventive aspect.

2. Clinical Varieties.—All the insanities of pregnancy may be said to show mental exhaustion in the early stage—a weariness of the rational functions and of the affections. In the insanity of pregnancy and in that of lactation the development is usually one of melancholia. In puerperal insanity the condition rapidly develops, as a rule, into an acute mania of the delirious type. Most cases show a well-marked perversion both of sexual desire and of the conjugal and maternal affections. One would expect that during pregnancy the capacity for sexual pleasure should diminish, but it is fortunately not normal that an intense loathing of the idea of sexual converse or a perverted appetite for vagrant indulgence should develop; yet these are common to pregnant women who become insane. Similarly, it is usual for the mother to turn against her husband or her child, and quite frequently she finds some fantastic substitute for the affection with which she has parted. Most cases are prone to impulses of a violent kind, and often towards those to whom she is normally most attached. There is nearly always confusion as to time and place and people, and a gradual loss of the sense of identities. Nearly every case manifests disturbances of sensory impressions, is beset by paræsthesias, or by illusions which to her disordered judgment seem fit reason for the delusions of misfortune or of grandeur which supervene.

(*a*) *The insanity of pregnancy* is the least frequent of the three varieties now considered. But, in my experience, many lactational cases have begun during pregnancy, and have merely been tided over. The seventh month is rather more critical than the others, and, if the patient goes past that, and if no septic development occurs in the puerperium, nothing serious may happen until well on in lactation. It may safely be said that all cases begin with mental depression, and the melancholia will continue and be a feature of the case unless some gross neglect of bodily functions, some sudden shock or some acute illness, precipitate the case and lead to mania. The usual case becomes indifferent and apathetic, and a considerable number become stuperose. A very large proportion become suicidal, and a considerable number have impulses of a homicidal nature. In nearly all melancholias of pregnancy the sensibility of the patient will be found reduced, and developments towards paræsthesias—perverted skin sensations, or tactual, or olfactory, or gross feelings of "internal disease"—are common.

(*b*) *Insanity of the Puerperium.*—This is perhaps the most dramatic, because the most sudden and demonstrative, of all insanities. After a difficult pregnancy and, from the obstetrical point of view, an unsatisfactory puerperium, the patient rapidly becomes delirious. Her appetite for food ceases, she passes an insufficient amount of urine, she becomes constipated, the lochia are inhibited, the patient has gone septic. It is not merely a local disorder: the whole system is obviously toxic. The mental accompaniments, after a more or less brief stage of depression, are that the mother ceases to be careful about her child, ignores her husband, is perverse in her temper, and more and more unmanageable, until manifest delirium with rise of temperature supervenes. The case rapidly develops, and takes on the characters of acute delirious mania, but with a shallower delirium, that is, a slighter oblivion, than in other comparable states. For example, the patient will dance, and sing, and shout, and her expressions of delight or of excited activity will increase in response to stimulation. She will probably recognise her husband and repel him; at the same time she will rush into the arms of her medical attendant or of a stranger. Her violence will be destructive, and she will throw her child across the room as readily as she will throw a cushion. No form of insanity better illustrates the evolution of insane energy; from the pulse, the appearance, the feel of the muscles, one would expect collapse; and yet the violent exertions continue. Then comes exhaustion, and the patient lies prostrate, roused only by fugitive pains or hallucinations, stimulated by vigorous attempts at feeding or by the necessities of nursing, sleeping fitfully or not at all, rambling in a muttering fashion if her voice is not too hoarse and her mouth not too dry for articulation. It may be some days before this stage

of prostration arrives, several days longer before recuperation begins, and it may be weeks before the patient is quiet and sensible; but the chances are that she will recover.

(*c*) *Insanity of Lactation.*—This form is commoner than the insanity of pregnancy, though it may be that the development of urgent symptoms has been postponed for months; and it is not so common as the insanity of the puerperium. It is an insanity of physical exhaustion, the result of continued depletion. It usually occurs in patients who have been suckling for more than six months. The typical variety is a slow melancholia with delusions, and with impulses often following obsession; but any grave occasion may determine an acute development either towards stupor or towards mania. Many cases are suicidal and many are infanticidal. From apathy about things in general the patient develops in the direction of neglect of her person, of the work of the household, and of the needs of her child. Her spirit is broken, her judgment lost, her self-control vanishing, and, if the disease be not arrested, she will suffer a deep melancholia, with gross delusions of a fearful kind and very dangerous impulses. I would draw attention to the physical state of these patients. It becomes a veritable cachexia. The pallid and perhaps icteric complexion, the sunken eyes and dilated pupils, the hollow cheeks, the wasted limbs and listless movements, suggest a pernicious anæmia, a diabetes, or an advanced phthisis. Yet in these cases, again, recovery is the rule.

It need hardly be said that departures from these types are very common. The last case that came under my observation was not at all like any of the varieties described. A young mother who had had a trying pregnancy, and who had suffered a severe shock towards the end of it, just succeeded in surviving puerperal delirium, but about the tenth day after delivery suffered a sudden impulse to injure her child with scissors, and then swooned. Thereafter she was beset for weeks with an obsession of blood; saw drops of it on every white surface she looked at, and, if her attention was not briskly employed, imagined patches or streams of it in front of her. She was separated from her child, carefully watched, generously fed, had tonics and open air exercise, and made an excellent recovery. She has had a second child without any untoward development. Such an exaggeration of the imaginative or delusional elements may occur in any of these insanities. Though melancholia is commoner, a pregnant insanity may go on to a deep stupor, or it may quite early develop into a mania. The same is true of lactational insanity, and especially of cases which occur in the early weeks, and which partake of the puerperal character. On the other hand, melancholia may persist as a sequel of puerperal mischief, or the patient may continue for weeks in a condition of mild mania characterised by delusions of grandeur.

3. Treatment, Management, and Prognosis.—These insanities are the most recoverable of all. The prognosis depends upon the heredity of the case, still more upon its history and especially upon the occurrence of previous attacks, most of all upon the management and treatment of the patient. But, in any case, 70 to 80 per cent of such cases recover. In the acute puerperal cases collapse and death are to be feared, while in the other varieties the fear is rather that the patient will live on into dementia.

As to treatment, I would emphasise the importance of prophylaxis; and that comes to be a question of the management of pregnancy and of the puerperium. I would remark that very few such good opportunities occur to the practitioner of earning the gratitude of his patient, as when he successfully pilots a tender woman of an insane predisposition through the physical and emotional distresses of child-bearing, and brings her safe into a sane motherhood. And I would submit, with deference, that both the general practitioner and the professed obstetrician might make more of such opportunities. It would be well if the practitioner, when he has booked a case for delivery at a specified date, when he has satisfied himself as to the pregnancy, when he has learned the history of previous confinements, if any, and when he has approximately gauged the diameters, it would be well if he would have minute regard to digestion and excretion in the case, to hygienic necessities, and would take reasonable precautions to preclude undue anxiety and worry. Morning sickness, for example, unless it actually threatens the patient's life, is still regarded as an incident hardly worth notice; constipation seems to be the rule; one rarely hears of the systematic dieting of pregnant women; and very few rules seem to be enjoined as to fresh air and exercise. Similarly, there is no instruction of the patient concerning the emotional and imaginative developments which she may expect; she is still allowed to believe that "maternal impressions" may have a serious effect upon her offspring; she is not warned that apprehensions and silly suspicions are sure to arise in her mind, and that she is to regard them as symptoms; and but little is done in the case of illegitimate pregnancies to moderate the rigours of a too severe public opinion. Prophylactic measures are especially called for in these days of city life. It might be an exaggeration to say that when a woman conceives she enters upon an abnormal state which requires a careful treatment, as if she had incurred a disease of nine months' inception; but it is true that, as women now live, pregnancy is a condition which calls for a most careful regulation and exercise

of all the bodily functions, and a corresponding attention to mental hygiene. There ought to be, throughout the pregnancy of a nervous woman, a scrupulous care of diet, periodic investigation into the condition of the alimentary tract, the liver, the kidneys, and the skin, and a systematic mode of life prescribed, which will ensure much more than the usual amount of fresh air, and not much less than the usual amount of recuperative exercise. If these precautions be taken, and dangerous developments corrected, even unlikely cases would be safely got through. Moreover, it is only by some such method, approaching that of the paternal type of family doctor, that the signs of incipient mental derangement can be observed and a grave development obviated.

If insanity of a fairly pronounced type has occurred, the first questions are those of management—where to treat the case, and in what kind of general conditions. It goes without saying that the patient must be as healthily conditioned as possible, that she must have as good air and as good food as circumstances permit. But she must also be carefully guarded. If her symptoms are aggravated by the life of her home she must be removed, though preferably not to an asylum. In any case she must have constant companionship; and if at home, the companion must not be one who will excite her jealousy, either concerning her husband or, if she has any, her other children. She must have long hours of rest in bed in a very airy and bright room, and she should, unless local conditions forbid it, have exercise in the open air, bedroom gymnastics, or massage.

In the insanity of the puerperium, and in that of lactation, the patient should be separated from her child and from her husband. Many puerperal cases lend themselves to home treatment. The conditions to be treated are the pelvic, the disordered digestion, and the mental excitability. Vaginal douching and uterine douching, when necessary, should be made use of as freely as if the patient were not insane, and her tendency to misinterpret these measures should be ignored. The mouth should be kept clean by the use of mild scrubbing with medicated vaseline, or an antiseptic wash. Food can be administered by the tube, and a little patience and care will usually induce tolerance of it, so that even the stomach may be washed out without greatly disturbing the patient. For the muscular excitement I believe in physical restraint—the packing of the patient for periods when she is supposed to rest in blankets bound round her trunk and limbs, and enclosed in a mattress tied round with straps or sheets. As to sedatives, it is the opinion of most that their use is inadvisable; but that is probably because of the unsatisfactory result of continued doses of too small a quantity. Several hours of sleep may be obtained by the rectal injection of paraldehyde in brandy with warm oil. Begin with a usual dose, but repeat and increase it, after a few hours, if the symptoms are favourable, giving three or four drachms in half an ounce of brandy, along with the oil, or with soup, or peptonised jelly. Similarly, and as an alternative, if the stomach tolerates liquid feeding, give bromide in full doses, working up to a drachm every four hours before the patient acquires a tolerance of it. But do not continue the use of a sedative for more than thirty-six or forty-eight hours.

For the insanity of lactation the treatment, again, is towards correcting the physical factors—by weaning the child, and building up the depleted tissues—removing the patient from the home environment which has exhausted her, and making herself, her husband, and her child safe from her violent impulses.

V.—SARCOMA-DECIDUO-CELLULARE OR DECIDUOMA MALIGNUM

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On the 16th July 1888, at a meeting of the Leipzig Obstetrical Society, Professor Sänger made a communication upon "two uncommon cases of abortion"; one of these cases was the first recorded example of the disease which we have now to consider. Since then nearly 100 cases have been recorded and a considerable literature has accumulated, from which we have learned that this disease differs sufficiently from all others hitherto described to justify its separate consideration. Observers are almost unanimous as to its connection with pregnancy, its clinical features, pathological appearances, and appropriate treatment, but they differ in their views as to its histogenesis.

The case reported by Sänger was so typical of the entire group of recorded cases, that I think a *résumé* of it will serve as an introduction better than any mere definition.

The patient was a young married woman, aged 23, who aborted at the eighth week in consequence of a fall from a railway carriage. The abortion was incomplete, and hæmorrhage continued for three weeks. In the fourth week the discharge became fœtid, and Sänger was called in. He found the patient very anæmic, with symptoms of septic absorption and putrefaction of the uterine contents. The uterus was thoroughly cleared out, hæmorrhage and discharge ceased, and the temperature became normal; the pulse, however, continued to be over 100, and recovery was so slow that she did not leave her bed for five months. Her protracted convalescence was attributed to a

small exudation in the left parametrium. An enlargement of the uterus was also observed, but was supposed to be due to subinvolution. She had not long left her bed when she was obliged to return to it again in consequence of fever and pains in the right hypogastrium, where a tumour as large as a goose egg had formed in the neighbourhood of the iliac crest. It was soft and tender, and, being supposed to be an abscess, was incised, but no pus escaped. It consisted of a soft spongy substance, of which about a handful was removed, leaving a cavity at the bottom of which lay the bare and roughened bone. On microscopic examination this tissue showed large multinucleated round cells, with spindle cells and numerous apoplexies. Soon after this operation pulmonary symptoms set in, with cough and dyspnœa, and she died seven months from the commencement of her illness.

On post-mortem examination the uterus was found to be infested with dark purple-red spongy nodules varying in size from that of a walnut to that of a large apple. The mucous membrane was smooth and nowhere perforated by the growths; metastases were found in the iliac fossa, lungs, diaphragm, and ribs. The left lung was compressed by a hæmatothorax; microscopic examination of the uterine tumours showed numerous apoplexies, large nucleated round cells, and cells resembling decidual giant cells, suggesting the idea that here was a new growth of the decidua belonging to the group sarcomata which had never before been described.

Clinical Features.—The clinical features of this disease are thus summed up by Sänger :—

1. A birth, abortion, or hydatidiform mole followed by—
2. Constant or repeated hæmorrhages.
3. After this, and generally following an examination or intra-uterine manipulation, putrid discharges with fever.
4. Increasing size and irregular shape of the uterus.
5. Rapidly progressing and towards the close intensely marked anæmia.
6. Recognisable metastases, especially in the vagina.
7. Cough, dyspnœa, bloody expectoration, and other symptoms of pulmonary metastases and hæmatothorax.
8. Rapid course of the disease, which usually proves fatal within six or seven months.

The first point of importance is the connection of this disease with pregnancy; in almost all recorded cases it has come on after an abortion hydatidiform mole, or very rarely after delivery at term. In about half the cases it has followed the expulsion of a hydatidiform mole—a remarkable fact of much importance with regard to its etiology. The second point is hæmorrhage, which either continues after the expulsion of the ovum or commences shortly afterwards. In a few cases it did not occur for a considerable time, and in one recorded by Whitridge Williams it was altogether absent, the first evidence of the disease being metastatic deposits in the vagina and lungs. In the majority of cases, however, it is the most prominent symptom, so that the patient soon becomes profoundly anæmic. The surface is pale, and in places slightly œdematous; the face especially assumes a pallid, icteric, cachectic appearance.

When the tumour sloughs the discharge becomes offensive and septic fever sets in. It is therefore of the utmost importance to observe the strictest antiseptic precautions in dealing with these cases.

On physical examination the uterus is found enlarged, often irregular in shape, and in some cases the os is sufficiently dilated to admit a finger, in which case the growth may be directly felt.

The course of the disease is one of marked malignancy; in fact, it is the most rapidly fatal of all malignant uterine growths, death occurring from hæmorrhage, septic infection, or pulmonary complications within a few weeks, rarely months, after the first symptoms have been observed.

Pathological Anatomy.—The uterus is usually found infested with new growths, varying in size and colour, but generally of a dark purple-red. They involve the muscular tissue of the uterus, so the peripheral margin is ill defined, and after removal the uterine wall appears thinned. The growth is soft and, where not coloured with blood, of a greyish-red hue; it closely resembles placental tissue in its irregular fissured surface, its fibrous appearance on section, extreme vascularity and numerous apoplexies, but it is much softer and more friable, and its uterine surface shows a remarkable tendency to slough.

Microscopically these growths are composed of a fibrous reticulum presenting an alveolar and cavernous structure containing numerous apoplexies, two varieties of cellular elements, and sometimes chorionic villi, but no glands, blood-vessels, or lymphatics. The two varieties of cells contrast remarkably. The one are large individual cells varying from round or polygonal to spindle shape, the nuclei are large, and the outline of their cell protoplasm is clearly defined; they resemble Langhans' cells. The others are protoplasmic masses of various shape which contain a number of irregularly shaped nuclei rich in chromatin, but with no line of separation between the cells, and resemble the syncytial layer which forms the superficial covering of the chorionic villi. Both varieties of elements show a marked tendency to retraction of their protoplasm and vacuolation; mitotic figures are frequently observed in the cells, but nowhere in the protoplasmic masses.

Metastases are generally found in the vagina, lungs, and other parts, and, as a rule, present

the same appearances as the primary disease; but Pick and Schmorl have shown that metastatic growths are not always malignant, and also that there have been cases in which typical chorio-epitheliomatous growths have been found in the vagina, lungs, liver, and other places, causing the death of the patient, and yet no primary uterine growths could be discovered (Schmorl, Schlagenhaufer, Schmidt).

Metastases in connection with such a malignant disease of the uterus are easily understood, but the occurrence of chorio-epitheliomatous growths in the vagina and elsewhere with a healthy uterus is not so readily explained. Schmorl, Kossman, and J. Nusmann supposed that there must have been a primary malignant disease of the ovum which caused metastases, and was subsequently expelled without infecting the uterus; but Pick and Gottschalk considered this most unlikely, and thought that more probably the metastases were originally of a benign nature, but afterwards underwent malignant degeneration. Schmidt considers that this hypothesis is borne out by the length of time that intervened between delivery and the appearance of the vaginal growths, viz., in Schmorl's case, eighteen weeks after delivery at term—in Schlagenhaufer's, nine months after an incomplete abortion—and in his own case, three months after expulsion of a hydatidiform mole.

Diagnosis. — An early diagnosis is of vital importance, and fortunately it is not difficult provided careful clinical and microscopic attention be devoted to it. When hæmorrhage occurs after an abortion or molar pregnancy the uterus should always be thoroughly evacuated by the finger and curette, so that one can be certain that everything has been removed and a smooth surface left. If after this hæmorrhage recurs, and a second evacuation reveals a quantity of soft friable tissue, the case is certainly not one of simple abortion, and is in all probability one of deciduoma; one must, however, be certain that the first curetting was thorough and complete. The size and shape of the uterus varies, but is never below the normal, and enlargement of the organ after discharge of the ovum is of much diagnostic value. The os is often sufficiently open to admit the finger, but may require artificial dilatation. Soft masses of friable material resembling placenta are then discovered, and portions removed by the curette should always be submitted to microscopic examination.

Treatment. — Total extirpation is the only method of treatment which has hitherto yielded any benefit, and it is of the utmost importance that this operation should be performed at the earliest possible time, before the patient has become exhausted by hæmorrhage, and before putrefaction sets in or metastases have occurred. It must not, however, be regarded as hopeless, even under such unfavourable conditions, for cases have been reported by Chrobak and Von Franke, where, in spite of evidences of pulmonary embolima, as shown by bloody expectoration and other symptoms, recovery has followed operation. Lönnberg-Mannheimer saw numerous metastases in the cervix, introitus vaginæ, and vagina, yet the patient had no return for eighteen months after operation. Cazin found a metatasis in the ovary; three years after the operation she was well. Schauta extirpated a metastatic growth from the vagina along with the uterus; at the time of publication there had been no return. Schlagenhaufer and Schmidt have reported cures after the extirpation of vaginal growths only—the healthy uterus being left.

Histogenesis.—According to most observers, this disease is always connected with and is probably due to pregnancy. The only dissentients from this view were Drs. Eden and Kanthack, who stated at the meeting of the London Obstetrical Society, in the discussion on Dr. Herbert Spencer's case, that the microscopic appearances were in no way peculiar, but similar to those found in sarcomata in other places, and that in some of the cases pregnancy was doubtful. In the discussion which followed Dr. Haultain's communication to the British Gynæcological Society, however, Dr. Eden stated that he was personally in full agreement with the views of J. Veit in the *Handbuch der Gynaekologie*, that the disease was a sarcoma modified by the occurrence of pregnancy. We may therefore assume as generally accepted that the disease is in some way due to pregnancy. In order to understand the conflicting views as to its origin we must briefly recall the normal condition of the tissues which it affects. The placental villi consist of a stroma covered by two layers of epithelium: an inner called Langhans' layer, composed of individual cells with a distinct cell wall, and an outer formed of protoplasmic masses without any line of demarcation, termed the syncytium. These villi penetrate the decidua serotina and maternal blood-vessels forming the placenta.

Now the disease which we are considering has been supposed by different observers to originate from each of these structures, and hence the varying nomenclature. Thus Sänger believes that it originated in the decidua, and he gave it at first the name of deciduoma malignum, but subsequently he preferred to call it sarcoma-deciduo-cellulare, because he believed that it originated in the cellular layer of that membrane after it had been altered by pregnancy. Gottschalk believed that it originated in a case described by him in the stroma of the villi. Marchand, Gebhard, and Haultain attributed its origin to the epithelium covering the villi, and Whitridge Williams to the outer layer or syncytium only, and termed it carcinoma syncytiale, chorion epithelioma, or blastoma-deciduo-chorion-cellulare.

These authorities may therefore be divided into those who hold that the disease is a sarcoma and those who regard it as a carcinoma. Sänger regarded it as a sarcoma of the cellular layer of the decidua because of the similarity of structure in both, the size and shape of the cells, and the presence of a reticulum, the only difference being that in the pathological cells the nuclei were larger and the surrounding protoplasmic ring narrower. He rejected Veit's theory that the disease was due to the modification of a pre-existing sarcoma, because no case had been recorded of the implantation of an ovum upon a sarcomatous or carcinomatous endometrium; and he believed that his claim to the growth being different from all others hitherto described stood or fell with its origin during pregnancy. Veit, however, in the newly published *Handbuch der Gynaekologie*, still maintains that the sarcoma precedes the pregnancy and is modified by it. He admits that no case has been recorded of the implantation of an ovum upon a carcinomatous endometrium, but in nodular sarcoma the membrane resembles that in myomatous uteri; and that as pregnancy occurs in the one, so it may also in the other. He considers that it is impossible to regard all the protoplasmic masses as true syncytium derived from the outer layer of the villi, but rather, as it has been shown, that in normal pregnancy other cells, such as the epithelium of the lining membrane of the uterus, and especially of its glands, often take on a syncytial form, and therefore syncytium in the wider sense cannot be regarded as a distinct tissue, but as a stage in the development of certain cells. So a sarcoma may under the influence of pregnancy come to resemble syncytium; and since fœtal elements are not always found in these cases, they cannot be regarded as the chief characteristic; but as masses resembling syncytium are the rule, the disease should be regarded as a process in which, under the influence of pregnancy, certain cells take on a syncytial character. Hydatidiform moles, so frequently associated with deciduoma, are not the cause but the result of uterine disease; and regarding deciduoma in a similar manner, the primary disease is to be sought for in the uterus.

In fibro-sarcomata the uterine mucous membrane is in a condition resembling that in myomata, in which pregnancy often occurs, and so it may in sarcomata; but under the influence of pregnancy the growth undergoes changes, and its cells assume a syncytial character. Under the influence of the growth, pregnancy may be disturbed or ended, a hydatidiform mole develop, or it may go to term. At the birth the entire ovum may be expelled, or a villus or villous epithelium retained grows into the veins and is carried into the circulation, causing metastases; in either case the tumour goes on growing and leads to a fatal termination. The disease remains the same whether it does or does not contain fœtal elements, the latter being accidental. The essentials are the original uterine disease and the occurrence of pregnancy, under the influence of which the syncytial changes in the growth occur.

Gottschalk, in 1892, described a case in which the stroma of the villi was changed into a structure formed entirely of large polymorphous cells, whose epithelial covering showed a remarkable proliferation; he regarded the disease as a sarcoma, and believed that it originated in the stroma of the villi. This case was the first in which the disease was diagnosed and the uterus extirpated during the patient's life.

The majority of pathologists regard this disease as of epithelial origin, and therefore a carcinoma; foremost amongst whom is Marchand, whose writings have done much to elucidate its pathology, and especially its connection with hydatidiform mole. In an elaborate paper, beautifully illustrated, which he read before the British Gynæcological Society in June 1899, Dr. Haultain warmly advocated Marchand's views, and stated that the chain of evidence in favour of this new growth being of epithelial origin had to his mind no weak link. The structural appearances, the physiological prototype of the young villi, and the interesting association with myxoma of the chorion, which it closely resembles anatomically, physiologically, and pathologically, all serve to prove that it is of epithelial origin.

It would be impossible in the present state of our knowledge to say which of these views is the correct one.

[Marchand's views are now generally accepted. It is believed that both the syncytium and Langhans' layer of cells are fœtal in origin, and that on this account the term deciduoma is incorrect. The name *chorio-epithelioma* is now generally accepted as more correctly indicating the nature and origin of this malignant growth. New problems have arisen in connection with this curious pathological development, viz. the relation to it of lutein cysts of the ovary, and the meaning of the occurrence of a similar growth in the male subject as a metastasis from a teratoma of the testicle.]

Pulex. *See* Parasites (*Insects, Diptera, Pulex irritans*); Skin Diseases of the Tropics (*Animal Parasites, Pulex penetrans*); Stinging Insects (*Pulex irritans*).

Pulna. *See* Balneology (*Austria-Hungary, Sulphated Waters*).

Pulmonary. — Belonging to the lungs (Lat. *pulmo*, a lung), *e.g. pulmonary embolism, pulmonary fibrosis, pulmonary tuberculosis*, etc.; or to the pulmonary valves of the heart, *e.g. pulmonary incompetence, pulmonary stenosis*, etc. *See* Lungs, Diseases of, and *Cross References. See*

also Breath (*In Pulmonary Gangrene*); Hæmoptysis; Heart, Congenital Malformations (*Pulmonary Atresia*, etc.); Heart, Myocardium and Endocardium (*Affections of Pulmonary Valves, Pulmonary Embolism*); etc.

Pulp. *See* Teeth (*Diseases of the Pulp*).

Pulpitis.—Inflammation of the tooth-pulp. *See* Teeth, Diseases (*Acute and Chronic Pulpitis*).

Pulsatilla.—A herb belonging to the Natural Order of the *Ranunculaceæ*, official in the U.S. Pharmacopœia; it contains an oil which has a poisonous crystalline principle, *anemonine* ($C_{15}H_{12}O_6$) and *anemonic acid* ($C_{15}H_{14}O_7$); pulsatilla has been used in the treatment of amenorrhœa, dysmenorrhœa, bronchitis, etc., in doses of from 2 to 3 grains of the powder.

Pulsation. *See* Pulse (*Heart Movements, Epigastric Pulsation*); Aneurysm (*Signs and Symptoms, Expansile Pulsation*); Aorta, Thoracic, Aneurysm and Dilatation.

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See also Aneurysm (*Arterio-venous Intercommunications, Symptoms*); Angina Pectoris (*Symptoms, Pulse*); Aorta, Aneurysm and Dilatation of (*Symptoms, Pulse*); Arteries, Diseases of (*Pulse in various Diseases*); Balneology (*Effects of Baths on Pulse*); Brain, Affections of Blood-Vessels (*Treatment of Cerebral Hæmorrhage, Prevent High Tension of Pulse*); Brain, Surgery of (*Concussion, Compression, Pulse*); Brain, Tumours of (*Symptoms, Pulse*); Children, Development of (*Clinical Examination, Pulse*); Chlorosis (*Symptoms, Pulse*); Climate (*Effect on Pulse*); Heart, Myocardium and Endocardium (*Physical Signs, Pulse in Aortic Stenosis, Aortic Incompetence, Mitral Stenosis*, etc.); Hypnotism (*Experimental, Phenomena, Changes in the Pulse*); Labour, Stages and Duration (*Second Stage, Constitutional Symptoms*); Lung, Tuberculosis of (*Complications, Circulatory*); Mediastinum (*Mediastinitis, Symptoms, Pulse*); Meningitis, Epidemic Cerebro-Spinal (*Symptoms, Pulse*); Meningitis, Tuberculous (*Symptoms, Pulse*); Nephritis (*Chronic, Circulatory Symptoms*); New-born Infant (*Physiological Peculiarities, Pulse*); Pericardium, Diseases of (*Acute Pericarditis, Pulse*); Peritoneum, Acute Peritonitis (*Symptoms, Pulse*); Physiology, Circulation; Pneumonia, Clinical (*Pulse*); Pregnancy, Diagnosis of (*Fœtal Pulse*); Puerperium, Physiology (*Pulse*); Scleremia Neonatorum (*Pulse*); Typhoid Fever (*Symptoms, Pulse*).

Introductory.—The older physicians placed great reliance upon the sensation communicated to the finger by the pulse. The Arabian and Moorish physicians recognised its importance, and our Celestial brethren pretended to be able to distinguish upwards of three thousand varieties of pulse. The Chinese even professed their ability to distinguish the sex of the fœtus *in utero* by feeling the pulse of the mother.

Before the introduction of the clinical thermometer the value of the pulse in diagnosis and prognosis was fully appreciated, and at the present day its examination is even more instructive, because the information obtained can often be usefully compared with the temperature.

In the present article it is proposed to consider some aspects of the subject which may prove of interest and value to the general practitioner.

It is a good rule to always make a point of feeling both radial pulses. The superficialis volæ branch may sometimes become the main trunk of the radial artery, and then the greatest pulsation will be found over the dorsal aspect of the thumb. When this abnormality exists there is a very feeble pulse in the usual situation, and this may mislead the unwary. A real difference in the radial pulse of the two sides may suggest the presence of an aneurysm or intra-thoracic tumour.

The Examination of the Pulse by the Finger.—There are a number of characters in the pulse which can be appreciated quite well without apparatus—others require instrumental investigation. When the radial pulse is felt, the physician first of all takes note of its *frequency*. But its *character* is of much greater significance. Thus the finger must try to gauge the volume of each pulse wave, whether it is *large* or *small*, and it is also important to form an impression as to the manner in which the artery is filled by the ventricular systole, whether *quickly* or *slowly*, and how the vessel wall collapses after impinging against the finger.

In addition, an estimate should be made of the *compressibility* of the pulse beneath the finger —whether it is soft or hard.

The *rhythm* of the pulse must always be noted, whether it is regular or irregular, and also the *character of the arterial walls*, whether they are thickened, atheromatous, or unduly contracted.

Frequency of the Pulse

The frequency of the pulse may be affected by many conditions which, either directly or indirectly, act upon the heart. To refer briefly to the *direct* causes which may influence the heart's action, mention may be made of the state of distension of the ventricles. If the heart is well filled it contracts better, though this is also determined partly by the state of the arterial resistance ahead. The frequency of the heart-beat is closely related to the condition of the arteries—dilatation of the vessels causes quickening of the heart, while their contraction may lead to marked slowing.

The rate of the beat is also affected in a striking manner—increased or slowed—by the character and quantity of the blood supplied to the heart muscle through the coronary arteries. It is highly important to bear this in mind, for the myocardium may suffer both from the effects of various toxins carried to it in the blood current, and also from a simple deficiency of blood. As is well known, the heart muscle may be profoundly affected by the toxins of many of the specific fevers, by anæmia, Bright's disease, gout, plumbism, alcohol, etc.; and, in pneumonia, muscle failure of the heart may result from the greatly lowered blood pressure, which interferes with the adequate blood supply to the cardiac walls through the coronary arteries.

Temperature may directly affect the heart's action. A poultice applied to the precordial region quickens the beat; an ice-bag slows it. The heart may, in a similar manner, be directly accelerated by the presence of hot liquids in the stomach, and also, perhaps, from direct pressure due to a distended stomach.

Lastly, among direct causes are the influences exerted over the heart by the vagus and sympathetic nerves.

There are a large number of *indirect* causes which affect the frequency of the pulse.[1]

Muscular exercise is one of the most conspicuous causes of increased pulse frequency. In some people the frequency may increase by 50 beats per minute with the exertion of running to catch a train. Violent dumb-bell exercise will produce the same result. The pulse-rate quickly returns to the normal in healthy persons after exercise, unless the active movements have been long continued.

The taking of food and drink—apart from stimulants—accelerates the pulse. Hot water, or more especially hot whisky-and-water, quickens the pulse, but a large draught of any cold liquid may slow it. It is interesting to note that, quite apart from the taking of food and drink, the movements of mastication, sucking, and swallowing produce a marked increase in the pulse-rate. Kronecker has shown that the act of swallowing seems to remove entirely the inhibitory action of the vagus upon the heart for the time being, and, according to Brunton, a glass of water slowly sipped may quicken the pulse-rate from 68 to 92 beats per minute.

Mental conditions and various emotional states affect the pulse-rate very considerably, either temporarily, *e.g.* when a healthy individual is being examined for life insurance, or more permanently, in persons who have sustained some severe shock to the nervous system, as from fright or grief.

The temperature of the skin indirectly modifies the pulse frequency in a reflex manner. The left ventricle responds very precisely to the call of the peripheral tissues for more blood. Thus in a heated room the peripheral vessels dilate, and the ventricle responds by beating more quickly; if they contract, as happens in a cold atmosphere, the heart-beat becomes less frequent. Clothing, by supplying warmth, increases the rate of the pulse; exposure of the naked body to the cold air slows it. The pulse-rate is found to increase in tropical climates, and is diminished in the Arctic regions.

The position of the body is an important matter with regard to pulse frequency. In recumbency the rate may become much slower than normal, and when a child is kept in bed for a considerable period the pulse may become remarkably slow and irregular. The same condition is found in children during sleep. The heart-beat varies a good deal according to the time of day. Its action is slowest very early in the morning, between two and four o'clock—this is the time when all the vital functions are at their lowest. It rises up to its maximum about ten or eleven in the morning, then comes down, but rises again between two and three o'clock in the afternoon, then keeps steadily going down again. This is the regular rhythmic way of the heart.

Change of position of the body—*e.g.* from the sitting to the erect posture—markedly affects the pulse-rate even in healthy persons, but the effect is not the same at all periods of the day. The greatest average difference occurs before noon. An increase of not more than 5 to 10 beats per minute is normal, and may be taken as evidence that the heart muscle is in

[1] For a full and connected account of the pulse as it is affected by age, sex, posture, exercise, food, and other natural causes, the reader is referred to an article by Dr. Guy in Todd's *Cyclopædia of Anatomy and Physiology*, vol. iv., 1847-49.

good condition, and that it is capable of responding adequately to a call for increased work should it be demanded. Many conditions, however, may lead to a contraction of what Mackenzie has called the "field of response" of the heart, and then the change of frequency between the sitting and erect positions may reach 30 to 50 beats per minute. In people of nervous temperament, and particularly in women, the pulse-beat may become exceedingly rapid on the slightest emotion or exertion. But for the same frequency of pulse, the effect of change of posture in the male is more than twice as great as in the female. The difference is still more strongly marked in early youth. During pregnancy the tendency to abnormally rapid action of the heart on change of posture, or with moderate exercise, is sometimes very evident, and would seem to indicate that if true hypertrophy of the heart has occurred it is certainly not of a compensatory nature (Figs. 1 and 2). This condition is usually a temporary one, but sometimes, after several pregnancies in rapid succession, the heart may be left with a permanently contracted field of response.



FIG. 1.—Tracing of the radial pulse from a healthy young primipara, 7½ months pregnant, in the sitting position.

Pyrexia tends to neutralise, and debility to exaggerate, this postural variation in the pulse-rate.



FIG. 2.—Tracing of the same pulse shortly after assuming the standing position. Shows increased rate of pulse and fall of blood pressure.

Age may be added as a cause which influences the frequency of the pulse. Thus during the first few weeks of life a pulse-rate varying from 120 to 150 may be regarded as normal. At this age posture seems to have little effect in modifying the rate. During the second year of life the rapidity of the pulse falls from 120 to 100 beats per minute. From the third year to puberty there is still a notable diminution in the pulse-rate, and after that it remains pretty constant till the age of sixty is reached. Then a quickening may commence which is often proportionate to the degree of arterial degeneration, and which depends also on the condition of the myocardium.

The pulse frequency varies with the sex of the individual, and is greater in women than in men. Still, a tall woman may have a slower pulse than a small man—the larger the frame the slower the pulse as a rule. The normal frequency in healthy people may vary very greatly, and constant rates of 40 and 100 have been noted in people apparently free from disease.

Significance of Frequency of the Pulse.—Mere frequency of the pulse, without regard to the state of the temperature or respiration, may have great significance, if it is constantly beyond what one recognises as normal. In uncompensated valvular disease the pulse is abnormally rapid even when the patient is recumbent. This is also the case in all forms of myocarditis, especially where the heart muscle has suffered from excess of alcohol, or from other toxic influences. The same condition of matters may follow overstrain, *e.g.* the "soldier's heart," and the so-called irritable heart. In neurasthenia, both in men and women, the pulse is sometimes persistently rapid, and is almost always very soft. In exophthalmic goitre the remarkable dilatation of the arterioles all over the body leads to greatly diminished peripheral resistance, and this may partly explain the abnormally rapid heart-beat. In puerperal insanity there may be much prognostic significance in the pulse-rate alone. Should a woman become maniacal a few days after delivery, without the pulse being greatly increased in frequency, there is little or no danger to her life, although she may lose her reason. A pulse of 120 to 150 at the onset of any form of puerperal mania is of almost invariably fatal significance.

A pulse ranging above 110, in a person older than twenty-five years, of the male sex, and not of nervous temperament, is, according to Copland, not without risk; the amount of risk will depend on concomitant circumstances. A pulse which is constantly of this rate may, under these conditions, be suggestive of latent tuberculosis.

Unusual slowness of the pulse may also attract attention to some morbid condition. A pulse of 50 or 60 or less may be normal to certain persons, and sometimes this feature belongs to several members of a family. The normal slow pulse is generally associated with a high arterial blood pressure, and the gouty diathesis may be the underlying cause. Those who are subject to gout, however, may have a pulse of high frequency.

Pressure on the brain may cause remarkable slowing of the pulse, especially when it affects the basal portions. Intense pain, *e.g.* that due to gall-stones, will often slow the pulse, and the blood pressure may be raised temporarily. Stokes has noted that in some cases of pericarditis the frequency of the pulse is diminished at the commencement of the disease. Structural lesions of the heart—fatty heart—may also give rise to very extreme states of slowness. Then there are many toxæmic conditions which lead to a slowing of the pulse-rate. On the invasion of certain specific fevers, and sometimes also before death in these cases, the pulse may

become greatly diminished in frequency. In Bright's disease the increased arterial pressure, which is excited by toxic influence, generally retards the heart's action; and in jaundice marked slowing of the pulse may occur. In children suffering from simple catarrhal jaundice, however, the pulse-rate does not often seem to be affected in this way. During pregnancy there may sometimes be a slowing of the pulse in the recumbent posture, but the acceleration caused by assuming the erect position is greater than it is under normal conditions. There is a toxæmia in pregnancy, and the *rate* of the pulse, in the sense that it reflects, more or less, the state of the blood pressure, is of some value as a symptom. The writer has made some observations on this matter, and believes that it is unusual to find either marked slowing of the pulse, or even much increase in arterial blood pressure, during the course of normal pregnancy. One might go the length of saying that the appearance of a strikingly *slow* pulse towards the end of gestation, if it persists for any length of time, forebodes danger. Examination of the urine may show that it is both scanty and albuminous, but the abnormal slowness of the pulse may precede the changes in the urine. For several weeks before the onset of eclampsia this pulse feature may be the only noteworthy symptom.

After the completion of the third stage of labour there is generally a marked fall in pulse-rate. The pulse may then become slower than that which is normal to the individual.

The pulse in children and also in young adults during convalescence from acute disease generally becomes very slow, and the individual beats are of unequal duration. The peculiar halting character of this slow pulse is perhaps specially well developed after the subsidence of the fever in cases of acute nephritis. It is also strikingly noticeable some days after the crisis of acute pneumonia, and may be regarded as a characteristic post-febrile feature of the pulse in children. In children who are apparently convalescent from diphtheria the pulse may sometimes become excessively slow and feeble, and heart failure from paralysis of the organ may then be imminent. Weber states that the pulse may fall to 28 beats per minute or even lower in these cases. In sclerema of the new-born infant the pulse may fall as low as 60 per minute.

Significance of the Pulse-rate when in Contradiction to the Temperature or Respiration.—It is always well to make a comparison of the pulse-frequency with the temperature chart, as this will sometimes lead to important conclusions. The significance of an elevated temperature without a corresponding increase in pulse-rate is of varied importance. In enteric fever the pulse is commonly between 80 and 100, when the temperature is considerably raised. This is to be regarded as a favourable sign. It is probable that a special toxic substance circulating in the blood is responsible for the relative slowness of the heart, and the combination is sometimes suggestive from a diagnostic point of view. In the early stages of influenza the same thing may be observed, and is of little moment, but occurring in the later stages it is a symptom of bad omen. In rheumatic fever it sometimes happens that a rapidly increasing temperature is not accompanied by a corresponding quickening of the pulse, and there may be delirium. Such a condition of matters would lead one to give a prognosis of the utmost gravity. Mention may here be made of a special circulatory feature in yellow fever, where there is a slowing of the pulse with a steady or even rising temperature. This peculiarity is believed to be of some value in the diagnosis of the disease, but seems to have no special prognostic import. In cases of cerebral compression from any cause, *e.g.* tubercular meningitis, the pulse is often unusually slow, and also irregular in rhythm. In cases of intracranial tumour and tubercular meningitis the mere slowness has little diagnostic importance—the chief significance lies in the fact that this feature coexists with marked elevation of the temperature. The contracted condition of the radial artery is a point of great importance in these cases.

While discussing the value of pulse symptoms, brief reference may be made to some of the conditions in which the pulse-rate is greatly accelerated without a corresponding rise of temperature.

Thus, at the commencement of scarlet fever a pulse of 140 to 150 may be found when the other symptoms are insignificant. This is always suggestive, for the toxin of the disease, in striking contrast to that of enteric fever, leads to a great acceleration of the heart-beat. The extraordinary acceleration of the pulse towards the close of tubercular meningitis is also noteworthy. In acute pneumonia an extremely rapid pulse at the onset, especially if it be notably soft, is almost always a fatal sign, even though the extent of lung involved is not large. It may indicate that the cardiac muscle has been completely disabled by the initial virulence of the toxin. When the pulse persistently increases in rate and becomes softer towards the end of the disease, the prognosis is invariably bad. In pulmonary phthisis, without marked pyrexia, a persistently rapid pulse is of serious import. In acute rheumatism a pulse of 120, without cardiac complications, signifies danger, whereas if pericarditis be present the pulse-rate may reach 150 per minute without serious results.

Every careful physician, if only for his own peace of mind, keeps an anxious watch upon the frequency of the pulse during the puer-

perium. At this time an increasing pulse-rate always excites greater concern than a rising temperature, and may be the precursor of septic infection. A high temperature during the puerperium need cause little anxiety unless the pulse becomes extremely soft and rapid.

After severe hæmorrhage—post-partum, from gastric or duodenal ulcer, etc.—the pulse-rate becomes greatly increased. But after forty-eight hours, or less, if considerable slowing has not occurred, the outlook is uniformly bad. The same remark applies to the frequency of the pulse after abdominal injuries and operations—a falling pulse-rate is always necessary to justify a favourable prognosis, even though the temperature is subnormal.

To sum up, then, it may be said that while undue slowing or great rapidity of the pulse may not, in themselves, suggest danger, still when viewed in conjunction with the temperature chart some significance is at times attachable. But the character of the pulse must always be taken into account. If it becomes more rapid and at the same time softer, it is a sign of approaching death.

The ratio of the pulse-rate to the respiration is sometimes suggestive. In pneumonia, according to the acuteness of the case, the pulse-respiration ratio is greatly altered, and in some cases of catarrhal pneumonia in children the ratio may be 1 to 2, or even 1 to 1·5. Notable perversion of the pulse-respiration ratio may be observed in some cases of fatty heart, where a pulse of 40 may co-exist with a respiration of 20 per minute, or a ratio of 2 : 1. In severe cases of rheumatic pericarditis, according to Walshe, the respiration rate may be greatly accelerated even though there be no pulmonary complication. Thus the pulse-respiration ratio may be 136 : 64 or 2·12 : 1.

Definition of the Pulse

The pulse consists in the sensation imparted to the finger when it is made to compress an artery.

It is necessary to discuss this definition in some detail, so as to clearly understand the nature of the pulse sensation.

The pulse-wave in the arterial system depends on the heart's action — on the left ventricle throwing a quantity of blood into the aorta. Briefly, we may put it in this way. Just before the aortic valves open, the pressure of blood in the left ventricle is the same as that in the aorta. After the aortic valves open, the pressure in the aorta gets increased by the systole of the ventricle, which adds several ounces to the bulk of blood already in it. This addition to the aortic contents leads to an *increased pressure* within the whole arterial tube. The radial artery, flattened under the finger or by the pad of the sphygmograph during the diastole of the heart, alters its shape under the distending force of the extra blood thrown into it by the ventricular systole. The pulse is therefore, essentially, the expression of *increased blood pressure.* By flattening the artery somewhat at a time when the resistance to compression is at its lowest, one more readily appreciates the change to the condition where the resistance to compression is at its highest. We purposely flatten the vessel against the underlying bone in order to observe this change of form, because it is then possible to appreciate the pulsation more efficiently. This is not essential, however, for the mere perception of the beat; for sometimes the pulse can be counted by simple contact with the finger.

In order to form a clear idea of what really constitutes the pulse, the writer knows of no more useful simile than that suggested by Sir William Broadbent. He says, "Such a pulsation can be felt on a large scale by placing the foot on the unyielding and inelastic leather hose of a fire-engine in action." The comparison is an excellent one, and it serves to contradict the impression that the pulse is due mainly to an *expansion* of the artery. The extra quantity of blood pumped into the aorta at the systole of the ventricle simply distends some of the larger vessels—any expansion of the smaller arteries is quite insignificant.

Again, the pulse, as it is felt in any superficial artery, *e.g.* the radial, is not due to the blood pumped into the aorta which has passed down there, but it is a wave, orginated by the impulse of the left ventricle, that has travelled along the particles of fluid in the artery. This pulse-wave, as it may be called, travels at a rate of 28 to 30 feet per second, while the rate of movement of the blood is only about 10 to 12 inches per second.

When a ligature is put upon an artery no pulse is felt beyond that point, but it is distinct up to the place where the artery is obstructed. This experiment enables one to appreciate the dependence of the pulse on the systole of the ventricle alone, when onward movement of the blood is prevented. The pulse is nothing else than the beats of the heart transmitted through the arteries.

Factors of the Pulse

The main factors concerned in the production of the pulse are :—

(1) The contraction of the left ventricle.
(2) The elasticity of the arterial walls.
(3) The resistance in the arterioles.

The character of the pulse may be influenced largely by the *left ventricle* in many ways. Roy and Adami consider that *the mode of contraction* of the left ventricle is a more important factor in the production of the different varieties of the pulse than the condition of the arterioles. The character of the systolic contraction depends partly upon the condition of the cardiac walls,

but it is also controlled in an important manner by means of nerves. The regulating influences of the vagus and accelerator must not be forgotten in this connection, and the intrinsic ganglia are equally important. Thus we may include the *nerves* of the heart among the factors of the pulse, and we find certain types of pulse irregularity which are due entirely to their effects.

The *state of the arterial walls* will clearly affect the character of the pulse, and this is important both with regard to degenerative or hypertrophic changes and also in relation to the condition of spasm, which may be excited by the abnormal contents of the vessels. Various states of toxæmia may induce this temporary condition of arterial spasm, which is accompanied by a general diminution in the calibre of the arteries during the time that the toxic substance is exerting its effects. Coincidently there is a marked alteration in the pulse. Among the factors of the pulse, then, we may also include the *blood*. Its impaired quality will lead to conditions of arterial spasm; and, if the toxæmia be long enough continued, a permanent alteration may be produced in the walls of the arteries.

The elasticity of the large arteries is a factor upon which the character of the pulse largely depends. By virtue of their elasticity the arteries always exert some compression on their contents, and this is so, even during the diastole of the ventricles. At each systole some distension of the larger vessels occurs, and thus the force of the heart is, so to speak, stored up, and its energy is gradually, instead of quickly, expended. It is otherwise when the vessels have become rigid from disease, whereby the functions of their elastic coats are seriously impaired. Thus the factors concerned in the production of the pulse in the child and in the old man respectively differ greatly, and for this reason one cannot compare the characters of the pulse at these periods of life. The inelastic artery of old age has a limited capacity of storing up the force of the left ventricle.

The state of the *arterioles* with regard to their permeability to the flow of blood must, of course, affect the character of the pulse by determining the rapidity of the outflow of blood from the larger arterial tubes. We also recognise that certain sections of the arteriole field may contract or dilate from various causes, and we must not forget that the vessels of the splanchnic area, by becoming fully dilated, may empty the arterial system and thus give rise to a pulse of extremely small volume at the wrist.

Therefore we cannot draw reliable deductions from the *volume* of a pulse, unless we take the condition of the other factors concerned fully into consideration.

Shortly before death, more especially in cases where the cardiac muscle is in good condition, one is repeatedly struck with the large volume, and sometimes with the good pressure, of the pulse. Such a pulse may mislead us as to the fatal issue. An abnormally relaxed state of the arteries along with a feeble left ventricle will give the same kind of impression to the finger as that given by a strong ventricle acting against a normal peripheral resistance. If the blood pressure be fairly good, we must remember that the dying heart is working at its highest pressure, while that of the strong, healthy individual has a considerable reserve power to fall back upon. In some instances the blood pressure maintained by the heart shortly before death may be said to be relatively high.

Examination of the Pulse by Means of the Sphygmograph

The only accurate method of recording the pulse is by means of the sphygmograph. Space will not allow of a description of the different forms of sphygmograph, and for a concise statement of these the reader is referred to Dudgeon's excellent treatise. Sphygmography has been in turn recklessly extolled and unduly depreciated. Those who assert that the sphygmograph is devoid of practical usefulness, and that it can be made to register any form of pulse-curve suitable to the occasion, simply exhibit their entire want of knowledge as to how the instrument should be employed. Such opinions belong either to those who are devoid of all mechanical skill, or to those who have not shown sufficient perseverance in mastering certain important details which can only be acquired after fairly long experience. With more perfect instruments the tracings would be more reliable, and probably a more skilled interpretation of the results is desirable. But the writer is convinced that much more can be learnt by the use of the sphygmograph than is generally believed. While the character of the pulse alone is not a safe guide to enable one to form conclusions in certain diseased conditions, still it will often afford indications of the utmost value bearing upon prognosis and treatment.

Dudgeon's sphygmograph is the one most in use in this country, and it is the only one which can be satisfactorily used in young children. If it be applied in the manner about to be described, most admirable and useful tracings can be obtained. The instrument must be a thoroughly good one; and I give this caution, because several which I have examined are of inferior workmanship, and quite unsuited for the purpose. The writing needle, which should be finely pointed, must swing evenly and easily, and the inelastic strap which fixes the instrument to the wrist must be replaced by pieces of elastic band. This simple modification immensely simplifies the process of obtaining successful tracings, for the instrument is very readily applied by tying the two pieces of

elastic, and the button can then be adjusted over the artery to a nicety.

Both practice and judgment are necessary to enable one to apply just the requisite degree of pressure to the spring of the sphygmograph to obtain a maximal swing of the needle, for if the pressure be too great the tracing will not be a reliable one. The pressure is applied to the artery after the instrument has been tied on to the wrist; and by watching the movement of the needle, one is able, after some experience, to judge when the swing appears to be greatest, and whether the button is lying accurately over the vessel. It is impossible to gauge the amount of pressure exerted by the button of the sphygmograph on the underlying artery and the tissues surrounding it, but this is not essential for our purpose. When the swing of the needle is maximal, it shows that the pressure which we have applied to the artery by means of the screw is the proper amount for the case under examination. We do not expect the reading on the dial to give any indication of the actual blood pressure.

But here it may be said that, after some experience in the use of the instrument, it is sometimes possible to judge in a general way of the blood pressure by observing the manner in which the writing needle responds to the turning of the screw. Thus, in recording a pulse where the blood pressure is undoubtedly high—*e.g.* acute nephritis—the needle will not begin to swing freely until considerable pressure is made on the spring by turning the screw to three or four ounces on the dial, and then the maximal swing may be obtained over a considerable range of pressure. Again, in a markedly soft pulse a good swing of the needle may be obtained after a very slight turn of the screw, and its excursion will noticeably diminish when too great pressure is applied in this way.

The pressure applied to the artery by the spring of the sphygmograph, in taking tracings from children under a year of age, need seldom exceed two ounces, and in new-born infants the index must often register the smallest possible degree of pressure, as even this is sometimes too great for the strength of the pulse-wave, and an incorrect tracing is obtained.

To get the best results, even the smoking of the papers is a matter of some importance, for if they are smoked too thickly the smaller elevations in the sphygmogram will not come out distinctly. They should be put into the tin holder and smoked lightly and evenly over a piece of burning camphor or an untrimmed oil-lamp.[1]

The writer may here draw attention to a small time-marker which he has attached to the Dudgeon's sphygmograph. A record of time is sometimes very important, and it is inadvisable to add anything to the bulk of the instrument if it can be avoided, especially when it is to be used in children. Von Jacquet's sphygmograph has a separate clockwork box to drive the time-marker, and it is a large heavy instrument. In the writer's arrangement the chronograph is worked from the large cog-wheel of the mechanism which drives the paper roller, and each tooth of the cog-wheel makes a division in the tracing which is approximately equal to one-fifth of a second.[1]

It is important to wind up the clockwork after each tracing is made, and also to make sure that the paper is being driven at a uniform speed.

The Arterial Blood Pressure

Definition.—The vascular system is over-filled, for were it not distended it could not contain all the blood. Thus the blood is lying in the vessels at some pressure, as is seen by the spurt which occurs when an artery is cut. The heart—one of the factors in the blood pressure—keeps up the over-filling of the vessels, and the resistance to the outflow of blood through the arterioles and capillaries determines the extent to which this over-filling occurs.

When an artery is compressed by the finger the state of distension of the vessel is appreciated; the *resistance to compression*, therefore, represents the blood pressure, and the *height* of the arterial pressure is determined largely by the ease with which the arterial over-filling is run off by the capillaries, that is, by the peripheral resistance. This is the other main factor in the blood pressure, and any variation in one or other of these factors may alter the pressure very considerably.

Relation to Ventricular Contraction.—The relation of increased heart force to blood pressure is not a simple one. When the ventricle contracts more powerfully the pressure is not always increased, for the heart's beat is always maximum; that is to say, the ventricles cannot, by contracting more forcibly, pump out more blood than they get from the auricles. The action of the auricles—"the feed-pumps of the ventricles"—is also important in relation to blood pressure, and great alterations may be produced by changes affecting the auricles alone. Increased work of the heart, however, without a coincident dilatation of the peripheral vessels, leads to a higher blood pressure.

Relation to Peripheral Resistance.—Changes in the peripheral resistance influence the height of the blood pressure enormously, and it is the state of contraction of the arterioles which mainly controls the extent of the resistance. Constriction or dilatation of the arterioles, dependent upon impulses through the vaso-motor

[1] White spirit-varnish (shellac dissolved in spirit) may be recommended to fix the smoked papers.

[1] This time-marker is fitted to the sphygmograph by Mr. W. M'Gregor, watchmaker, Castle Street, Edinburgh.

nerves, will increase or decrease the pressure. The vessels of the arteriole and capillary fields may contract or dilate in sections from local and other causes. As Brunton has pointed out, there are three channels through which the blood finds its way from the arteries into the veins. There are the vessels of the skin, the vessels of the intestine, and the vessels of the muscles. Little attention has been paid, hitherto, to the vessels of the muscles in this connection, but according to Brunton, the outflow through these is equivalent to that of the other two channels together. The important point with regard to the blood pressure is, that although the vessels of the skin and intestine are contracted, yet the peripheral resistance may not be much increased, and a higher blood pressure may not result; the blood still escapes freely through the vessels of the muscles. Vaso-constriction causes the left ventricle to contract more powerfully, and tends to slow the pulse; a slow pulse, therefore, generally indicates a higher blood pressure than a fast pulse. Dilatation of the vessels causes a quickening of the heart, and this, according to the researches of MacWilliam, is almost always due to the suspension of vagus activity; it is very doubtful whether the same result is ever dependent upon stimulation of the sympathetic.

Relation to Arterial Walls.—From what has been already said it will be seen that the relation of the blood pressure to the state of the arterial walls is a very complex one. The radial pressure may be considerably increased both when the vessel walls are felt to be tightly contracted, and also when the artery is large and relaxed. In the one case there is a contraction of the muscular coats of the arterioles and larger arteries from direct or indirect irritation of the vaso-motor centre. This condition is typically present in some cases of tubercular meningitis and cerebral tumour, and it may also be induced reflexly by severe abdominal pain, tympanites following a laparotomy, and profound shock. This kind of arterial spasm may sometimes be unilateral, in neuralgias, lead colic, etc. In the other case the radial blood pressure is raised by increased peripheral resistance mainly confined to the capillaries. The artery is then large, and the pulse may be full and bounding. Obstruction of the capillaries, due to various toxins in the blood, is the commonest and most persistent cause of high blood pressure. Sometimes when the pulse feels small and weak, owing to involvement of the radial artery in the area of increased peripheral resistance, the blood pressure in this vessel may not be increased, and yet, as Oliver points out, it may be greatly raised in the central arteries.

When the arterial walls are thickened and sclerosed, the blood pressure is not always raised. The calibre of the vessel may be increased or diminished, but the blood pressure may, in either case, be high or low. In the young the elastic wall of the artery plays an important part in maintaining a good blood pressure, and as age advances the resiliency of the vessel is largely impaired. In very old people the contracted "field of response" of the heart is a factor which must be taken fully into account; for this reason a low blood pressure may be present when the radial artery is hard and cord-like.

In forming an estimate of the blood pressure the important point to note is the *compressibility* of the vessel—the force necessary to obliterate the pulse-wave. In advanced life the thickened condition of the radial walls may lead one to call the pulse one of "high tension," but, in some cases, from the ease with which the finger can obliterate the pulse, we judge the actual blood pressure to be low.

Relation to Pulse-Rate.—Marey has shown that the blood pressure controls the rate of the heart's action—the pulse-rate varies inversely as the arterial pressure. In normal conditions high pressure in the vessels slows the pulse, and a low pressure quickens it, but sometimes the important factor in maintaining a good pressure in the arteries is the *increased rate* of the pulse. Thus in the infant the heart's action is exceedingly rapid, and only a small quantity of blood is thrown into the aorta by each ventricular contraction. The pulse at this age is of small volume, but it is one of relatively high pressure. At this age an adequate pressure is maintained by a series of rapid and weak systoles; in later life the same result is attained by slower and more powerful ventricular contractions.

Relation to Volume of Blood in the Circulation.—The total volume of blood in the circulation is another important factor in the blood pressure, and every change by abstraction or addition alters the pressure. But the alterations produced on the circulation by venesection or transfusion are of a very temporary kind. Hæmorrhage up to 2 per cent or 3 per cent of an animal's weight scarcely affects the blood pressure at all, but up to 4 per cent or 6 per cent of the body weight the pressure may temporarily fall to nearly zero. After severe hæmorrhages an average pressure is soon restored by increased absorption of fluids from the tissues into the circulation, and though transfusion may raise the blood pressure for a time, and thus tide a patient over a period of great danger, the increased pressure is not maintained. In certain toxæmic conditions, indeed, where the blood pressure is abnormally high owing to extensive arterial spasm, as occurs in some cases of puerperal eclampsia, the writer has shown that one of the early effects of adding to the fluid in the circulation, by giving large saline infusions, is to lower the blood pressure remarkably (Figs. 4 and 5). This effect is probably brought about by the dilution of a toxin,

which, by inducing vaso-constriction, greatly increases the peripheral resistance. Venesection, even when freely practised, will not permanently reduce the general arterial pressure, but as



FIG. 4.—Radial sphygmogram from a young primipara suffering from puerperal eclampsia. Slow pulse of high pressure. Vessel wall markedly contracted.

a temporary expedient, and particularly as a means of relieving the heart and lungs, it is of very great value.

Relation to Composition of the Blood.—Wrong composition of the blood, as well as alterations

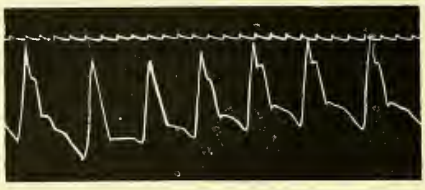


FIG. 5.—Tracing from the same patient shortly after an infra-mammary infusion of one pint of saline solution. Quicker pulse of diminished pressure with relaxation of arterial walls (enlarged calibre).

in the specific gravity of the plasma, by unsettling the regular onflow of the corpuscles through the capillaries, will increase the peripheral resistance and raise the blood pressure, *e.g.* Bright's disease, anæmia, etc.

Relation to Respiration.—Respiration acts on the blood pressure in two ways. Generally speaking, inspiration lowers the pressure, while expiration increases it, but with a high arterial pressure these effects may be very slight.

Measurement of the Arterial Pressure in Man

The Mean Arterial Pressure.—The educated finger can recognise when the arterial pressure is unduly high or abnormally low, but in health there is a certain pressure maintained in arteries as a resultant of the factors already mentioned.

Hamilton expresses it tersely thus: he says—"Were the heart to cease beating, the aortic valves to be closed, and the capillary outlets to be occluded simultaneously, the pressure of the blood within the arteries would represent this mean pressure." The intra-arterial blood pressure varies between two extremes—the maximum, which occurs during the ventricular systole, and the minimum, which is present between the pulsations, during the ventricular diastole. The relations which these two pressures bear to each other may vary greatly. In cases of high blood pressure, *e.g.* Bright's disease, the systolic and diastolic pressures approximate; while some cases of low blood pressure, *e.g.* aortic regurgitation, show greatly increased pressure during the ventricular systole, and a great diminution during the diastole. The terms "minimum pressure" and "mean pressure" are sometimes used synonymously, but others place the mean pressure at some point between the diastolic and systolic pressures.

Methods of Estimating the Blood Pressure.—There are at the present day many instruments—sphygmometers—which are used to measure the blood pressure in the human subject. There are two methods by which this may be attempted, and two distinct principles are involved. Some of the instruments profess to gauge the pressure necessary to obliterate the pulse; this is the idea underlying the method of von Basch, and it is intended to register the maximum or systolic blood pressure. Other forms of sphygmometer are based upon a principle suggested by Marey, which consists in increasing the pressure on the outside of an artery until the pulse-wave attains its greatest amplitude. This method was originally adopted by Mosso, and all the instruments of recent introduction make use of this principle. It would seem probable that this method, when properly carried out, must give the minimum or diastolic pressure; for experimental proof that this is so the reader is referred to an excellent article by Drs. Howell and Brush (*Boston Med. and Surg. Journ.*, vol. cxlv. No. 6). This, however, is not the invariable view taken, for in the Hill-Barnard instrument, which adopts this principle, the maximal pulsation, according to Hill, indicates the mean arterial pressure and not the diastolic.

The method of determining the arterial pressure in man by obliterating the pulse (von Basch, Potain, and others) should give the systolic or maximum pressure in the artery examined. But all the instruments on this principle really give a supramaximal reading, and are seldom employed.

Of instruments constructed on the other principle—that of increasing the pressure on the outside of the artery till it equals the pressure within—there are many forms. One of the simplest has been introduced by Hill and Barnard, and the writer here quotes Hill's description of the method and results from Halliburton's *Physiology* (4th ed.):—

"The instrument consists of a vertical glass tube about 5 inches in length, which expands above into a small bulb, and is closed at the top by a glass tap.

"A small india-rubber bag is fixed to the tube below; this is surrounded by a metal cup, attached in such a way that only the base of the bag is exposed. The bag is filled with coloured fluid. On pressing the instrument down over the radial or other artery the fluid rises in the tube and compresses the air in the bulb; the

air acts as an elastic spring. The more one presses the more the fluid rises; at a certain height the meniscus of the fluid exhibits more pulsation than it does at any other height (*maximal pulsation*). The tube is empirically graduated in divisions that correspond to millimetres of mercury pressure. The point of maximal pulsation gives the arterial pressure. Before each observation the tap is opened, and by gentle pressure on the bag the fluid is set at the zero mark on the scale. Thus errors due to changes in barometric pressure or temperature are avoided.

"We now come to the explanation why the maximal pulsation gives us a reading of arterial pressure. If the mean pressure inside and outside an artery be made equal, then the wall of the vessel is able to vibrate at each pulse with the greatest freedom. The mean pressure is less than the systolic, but greater than the diastolic pressure; thus during the heart's systole the artery is opened out to its fullest extent, while during the heart's diastole its lumen is obliterated; hence the vessel wall swings with the greatest amplitude. If the pressure exerted by the sphygmometer is less than the mean arterial pressure, the artery will not be compressed to its utmost during diastole; if, on the other hand, the pressure exerted is greater than the mean, the artery will not fully expand during systole. In either case the pulsation will not be so great as when the pressure exerted on the outside of the artery equals the mean pressure within.

"By recording the arterial pressure in the dog with a mercury manometer, and at the same time taking readings with the sphygmometer, the instrument has been proved to give accurate results."

Hill and Barnard have invented a larger instrument on the same principle, which takes the pressure in the large arteries of the arm just above the elbow. A thin india-rubber bag, fixed inside a leather armlet, is strapped round the arm. The india-rubber bag is connected by a T-tube with an air-pump and a spring manometer (Fig. 7). The pressure in the bag is then raised until the needle of the manometer gives its maximal pulsation, when the mean pressure is accurately registered. There are many obvious fallacies in the results obtained with this instrument; the elastic walls of the bag and tubing, the air conduction to the recording apparatus, the muscular development of the arm, all modifying the results.

In recording the blood pressure with either of these instruments it is sometimes difficult to determine the point at which maximal pulsation occurs. Thus the movement may be large between 140 and 150 mm. on the scale, may become distinctly less at 130 mm., and may then appear as great as before at 120 mm. Which reading in these cases indicates the mean arterial pressure? But the maximal pulsation can often be readily noted, and then a definite reading is obtained. While the writer does not think that this reading can always be taken as representing the *mean* arterial pressure, still in a given case he believes it indicates the general state of the pressure. For comparative purposes this would seem to be sufficient, and the variations in the readings from day to day will furnish valuable information; the method used gives reliable results when a series of observations are made on the same patient.

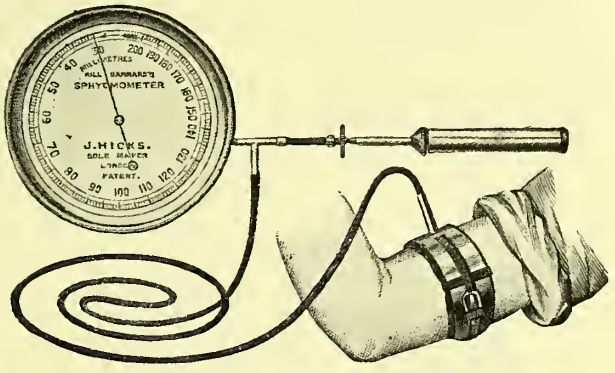


FIG. 7.—Hill and Barnard's Sphygmometer.

According to Hill's observations the normal pressure in the radial artery in healthy young adults is 110 to 120 mm. of Hg, and it appears to be as constant as the body temperature.

Dr. George Oliver has devoted much time to the study of the normal variations of the blood pressure under various physiological conditions, and also to certain abnormal variations produced by massage, cold baths, etc. He has contributed largely to our knowledge on these subjects, but in this place we shall only refer briefly to new instrumental methods of observation introduced by him.

To overcome the manifest difficulties encountered in determining the blood pressure in man, Dr. Oliver has shown very great ingenuity and originality. He has carefully considered the matter from every point of view, and has arrived at the following conclusions:—

(1) That the blood pressure in man can be correctly estimated through a fluid medium, but not through a solid one.

(2) That variations in the resistance of the tissues do not vitiate the correct reading of the mean arterial blood pressure.

(3) That variations in the calibre of the arteries do not disturb the reading of the mean arterial pressure.

(4) The adoption of the fluid principle obviates the errors due to the malposition of the solid pad and to abnormal conformation.

The Hæmodynamometer, or Blood-pressure

Gauge.—Dr. Oliver's hæmodynamometer, or blood-pressure gauge (Fig. 8), consists of two main parts—(1) the pad; and (2) the recorder. The pad is made of thin rubber, and is filled with water containing a little glycerine. It is placed over the radial artery and transmits the pulsations of the vessel to the recording apparatus.

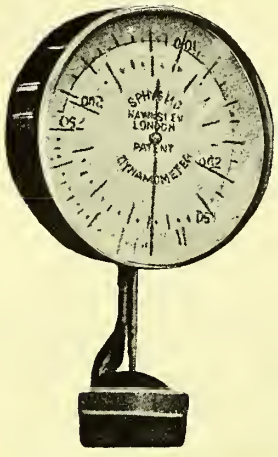


FIG. 8.—Oliver's Hæmodynamometer.

The principle followed is to equilibrate the fluid pressure within the pad to that within the artery, so that when that point is reached, the two pressures being exactly balanced, as it were, and the pad then being but a diverticulum of the artery, the maximum motion of the pulse-wave is developed. In principle it is identical with the sphygmometer of Hill and Barnard, but it is a more convenient form for clinical use.

We have tested both these instruments together on a series of patients in order to ascertain how the readings would compare in estimating a blood pressure. With Dr. Oliver's instrument a much higher mean arterial pressure was invariably indicated. We are therefore of opinion that there is still no ready and accurate means of estimating the arterial blood pressure in man. Dr. Oliver's observations with his hæmodynamometer are reliable, so far as the instrument determines, and in his hands supplies valuable information; but what we wish to emphasise is that there is, as yet, no means available to the general practitioner of estimating the blood pressure clinically in a manner at all comparable to taking the temperature.

The Arteriometer.—The other instrument devised by Dr. Oliver—the arteriometer—is used to measure the calibre of any superficial artery (Fig. 9). Two conditions must be observed in order to make the reading as reliable as possible—(1) the wrist must always be extended at a uniform angle; and (2) the same part of the artery must be observed on all occasions.

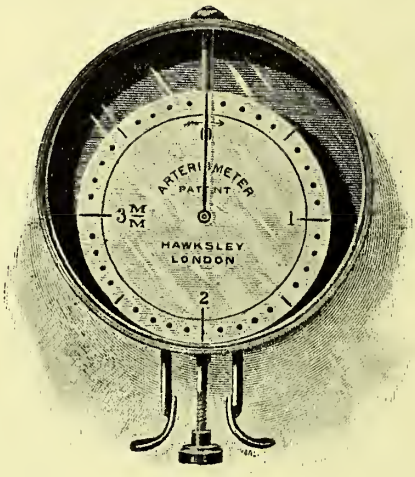


FIG. 9.—The Arteriometer (Oliver's).

We have a high opinion of the value and scope of usefulness of this little instrument, have used it frequently, and have found it to indicate a diminution of the radial calibre when the finger judged the artery to be contracted. Dr. Oliver has made very numerous observations on the calibration of the arteries in health and disease, and some of his results are extremely suggestive.

THE INTERPRETATION OF THE SPHYGMOGRAM

The Normal Syhygmographic Tracing.—Fig. 10 will serve to show the various parts of a typical sphygmogram from the radial artery. Certain points may be noted: the up-stroke (*a*), the apex of the tracing (*b*), the beginning of the down-stroke (*c*), the tidal or predicrotic wave (*d*), the dicrotic or aortic notch (*e*), the dicrotic wave (*f*), and, lastly, the remainder of the down-stroke. At the point *g* there is sometimes a second elevation similar to the dicrotic

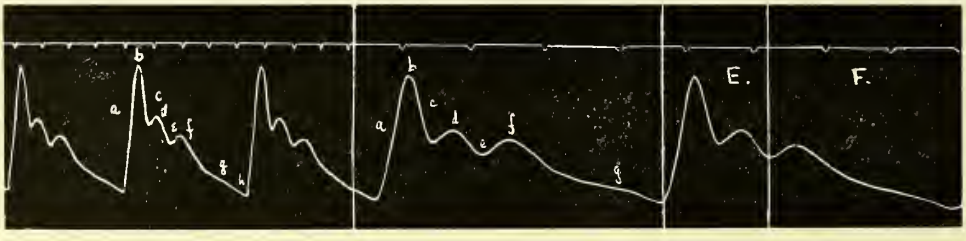


FIG. 10.—Normal sphygmographic tracing. *a*, The up-stroke; *b*, the apex of tracing; *c*, the down-stroke, which may be of varying length; *d*, the so-called tidal or predicrotic wave; *e*, the dicrotic or aortic notch; *f*, the dicrotic wave; *g*, the post-dicrotic wave; *h*, termination of down-stroke. E, the systolic portion of the pulse-curve—the "*systolic wave.*" F, the diastolic portion of the pulse-curve.

wave, but smaller—the post-dicrotic wave (Fig. 11). At *h* the down-stroke ends; and, according to some, that portion between *g* and *h* indicates the period of rest in anticipation of the ventricular contraction. The lowest point of the



FIG. 11.—Radial sphygmogram showing the post-dicrotic wave.

tracing has been called the base line by Landois, and the respiratory line by Mahomed. Its level may vary with the movements of respiration, particularly when there is great dyspnœa.

While all observers are agreed upon the main features of the sphygmographic tracing as above indicated, much difference of opinion still exists as to the mode of production of the various events, and as to their individual variations. We may shortly review some of the interpretations put upon the pulse-curve.

The up-stroke of the needle means a rise of arterial pressure, and it is commonly called the percussion stroke, because it is usually produced by the shock received by the blood column on the sudden bursting open of the aortic valves, which is transmitted almost instantaneously throughout the whole arterial system. Then with the commencement of the down-stroke a fall in blood pressure is indicated, and a rounded or pointed wave (*a*, *b*, *c*) is formed. This portion of the tracing is called the *percussion wave* by some, and includes the so-called percusssion stroke. The next feature is the *tidal* or *predicrotic wave* (*d*). Some writers explain it in this way: when the pressure in the artery begins to fall, the decline of the needle is delayed by a brief period of maintained expansion, which may amount to an actual wave of renewed expansion. This elevation is much more marked in tracings made from the carotid and brachial arteries, *i.e.* nearer the heart. The instrument exaggerates this feature of the sphygmogram.

The next event, however, the *dicrotic wave* (*f*), corresponds to a true secondary wave of increased blood pressure in the arterial system. The *dicrotic notch* (*e*) marks very precisely the time of closure of the aortic valves.

After these general statements it is necessary to discuss some of the features of the pulse-curve more particularly. It may be said at once that much confusion has arisen from the use of the terms "percussion wave" and "tidal wave" in describing the sphygmogram. It is admitted by all that these two waves occur during the systolic contraction of the ventricle, and that the other waves occur during the diastole. Yet the percussion wave is usually regarded as a distinct event in the pulse-curve, which should be entirely separated from the tidal wave except under certain conditions where the two waves are blended to form a single summit to the tracing. Mahomed regarded the systolic portion of the pulse-curve as composed of two factors, *i.e.* the percussion and the tidal waves. The percussion wave he described as the sudden primary expansion of the artery due to the shock of systole on the aortic contents; the tidal wave he supposed to be due to the more gradual distension of the artery. Burdon Sanderson says: "As a rule, the sensibility of the finger is most affected at the moment the artery attains its acme of tension, *e.g.* at the height of the tidal wave, but in low-tension pulses the same cause which jerks up the lever of the sphygmograph (percussion stroke) communicates the first sensation to the finger." It will be instructive to quote the following passage, from the writings of Mahomed, bearing on the significance of the tidal wave. He said: "The simplest pulsatile movement that can be conceived in an elastic tube is the mere passage of a wave of fluid through it, causing more or less sudden expansion and a gradual collapse of the tube, as it passes through it; such a wave is the foundation of the pulse, and has been called the 'tidal' wave. If the impulse imparted to the fluid is more sudden, an element of percussion or shock will be introduced, giving an abrupt and vertical up-stroke, from the jerking up of the lever by the sudden expansion of the artery. Owing to its acquired velocity, this movement of the lever is rather greater than the corresponding movement in the arterial wall which produced it, and on reaching its highest point it falls suddenly by its own weight, till it is again caught and perhaps slightly raised by the tidal wave, which is now only reaching its maximum of distension. . . . The tidal wave is the true pulse-wave, and indicates the passage of a volume of blood through the arteries, pumped into them by each contraction of the heart. It resembles the passage of the tidal wave or 'bore' up a river; hence its name. It is transmitted more slowly than the percussion wave, or rather attains its maximum intensity more gradually; hence their separation in the tracing. Though they usually commence to distend the artery together, the percussion wave necessarily attains its maximum intensity instantaneously, it being only a shock, while the tidal wave does so more gradually. Sometimes a considerable interval elapses between them. Frequently they are inseparable, the percussion wave not existing or else being merged into the tidal."

Although Mahomed here makes use of the terms "percussion" and "tidal" waves, still he appreciates the important fact that the

former is of instrumental origin when it appears as a separate apex to the pulse-curve. From this description we are led to a true conception of the systolic portion of the sphygmogram.

The Systolic Wave.—From a study of simultaneous tracings of the pulse, aortic pressure, apex-beat, and intra-ventricular pressure, as worked out experimentally by Hurthle, von Frey, and others, it is clear that the up-stroke of the sphygmogram coincides exactly with the rise of the aortic pressure—the so-called percussion wave does not occur earlier than this, and therefore it must not be regarded as a separate event in the tracing (Fig. 12). Dr. James Mackenzie has greatly simplified matters by applying the term *systolic wave* to the first part of the sphygmogram, from the commencement of the rise to the dicrotic notch. This, in the writer's opinion, is the most correct and rational method of interpretation yet adopted, and it indicates the true relationship of the ventricular and arterial pressures.

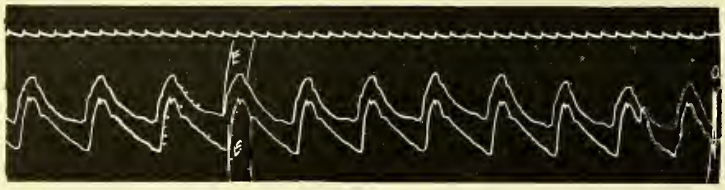


FIG. 12.—Simultaneous tracings from the aorta (fusiform dilatation) and radial pulse. The spaces E represent the duration of the ventricular outflow; the up-stroke of the sphygmogram corresponds exactly with the rise of the aortic pressure. (Time register in fifths of a second—writer's time-marker.)



FIG. 13.—The systolic and dicrotic waves—the true intra-arterial waves.

The principal fact to lay hold of is this, that there is only a *single* wave movement produced in the arterial system at the time the ventricular contents are forced through the aortic valves, and this, as Mackenzie suggests, may be called the systolic wave.

In the tracing of a low-pressure pulse (Fig. 13), the heart movement which gives rise to this wave is apparent as a single elevation, and then the second and slighter increase in arterial pressure which follows is depicted as the dicrotic wave. Therefore, when the radial artery is compressed by the pad of the sphygmograph, these two waves—true intra-arterial waves of pressure—produce two expansions of the vessel, and the simplest type of sphygmogram is represented.

The Dicrotic Wave. — With regard to the nature of the dicrotic wave we may give the following explanation :—After the completion of its systolic contraction the ventricle begins to relax, and in doing so any support afforded to the aortic cusps by its contracted walls is withdrawn. A negative wave is thus set up which travels towards the heart. This wave is arrested by meeting the closed aortic valve, and is reflected as a positive wave towards the periphery. Hence it is accompanied by a renewed rise in the blood pressure, and is indeed sometimes appreciated by the finger as a second though feebler pulse-wave.

That the two waves—systolic and dicrotic—are the only essential ones in a pulse-tracing is easily demonstrated by allowing the blood to spurt from a cut artery on to the surface of a revolving drum, when the so-called hæm-autograph is obtained.

Modifications of the Systolic Wave.—It so happens that what is generally described as the normal sphygmographic tracing shows more than these two waves, for between the summit of the first wave and the dicrotic notch there is a second upheaval (Fig. 10). This upheaval may sometimes rise to a higher level than the first summit, or it may be poorly represented. The systolic wave may therefore appear in the sphygmogram as a single curve, or it may be broken up into two or more distinct elevations owing to the participation of other factors. The sphygmograph plays an important part in the breaking up of the systolic wave, but the arrangement of the two summits—*i.e.* the time of occurrence of the second wave—is determined largely by the degree of obstruction to the outflow of blood from the ventricle into the aorta, and also by the state of the resistance in the peripheral arteries. Thus interference with the propagation of the systolic wave from marked narrowing of the aortic orifice will produce a fairly constant and characteristic modification of the summit of the sphygmogram —the wave is broken up early, and the second elevation, which may be pointed or rounded, forms the highest part of the tracing (Fig. 14).



FIG. 14.—The modification of the systolic wave in aortic stenosis—the second summit forms the highest part of the tracing.

Again, with the normal propagation of the

systolic wave a contracted state of the peripheral arteries may also lead to a fairly typical division of the summit. Both waves may be of about equal size, with a notch of varying depth between them (Fig. 15). With greater peripheral resistance the tracing is frequently of the type shown in Fig. 16, and should the resistance become still further increased the pulse may become anacrotic (*see* p. 399). Under certain conditions, *e.g.* pyrexia, where the peripheral vessels are dilated and the ventricles excited, a somewhat similar summit may be formed, and is entirely due to instrumental defects. In some cases of aortic regurgitation without stenosis, this kind of summit is frequently seen (Fig. 17).

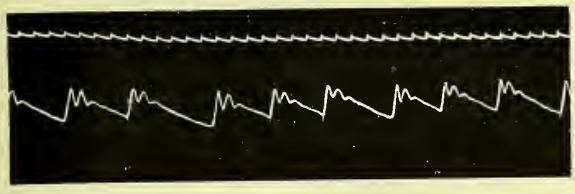


FIG. 15.—Modification of the systolic wave produced by moderate peripheral resistance. There are two summits of about equal size with a notch between them. (Tubercular meningitis—child æt. 5 years.) The type of pulse irregularity is also shown.

The manner in which the systolic wave is broken up is in many cases suggestive, but the writer agrees with Mackenzie that it is better to discard all terms of signification in naming the various summits. There is the single heart wave—the systolic wave—and the dicrotic wave, which is dependent upon the closure of the aortic valves. Modifications in the outline of this simple form of sphygmogram may result from the participation of the other factors mentioned.

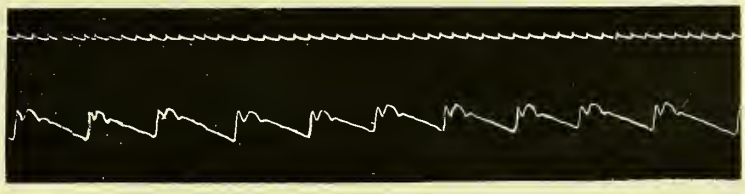


FIG. 16.—Modification of the systolic wave produced by pronounced increase in peripheral resistance. (Tubercular meningitis—child æt. 3¼ years.)

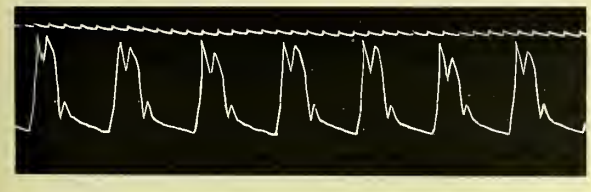


FIG. 17.—Pulse in aortic regurgitation without stenosis. The double summit of the systolic wave is of instrumental origin.

Views of Roy and Adami.—The views of Roy and Adami as to the origin of the leading features of the systolic portion of the pulse-tracing may be briefly referred to; but, for the reasons in favour of such changes in nomenclature as they suggest, their paper must be consulted (*Practitioner*, 1890). Roy and Adami lay great stress upon the mode of cardiac contraction, and, generally speaking, they recognise, in addition to inertia waves, *three* heart waves. The up-stroke corresponds to a primary heart wave, and is due to the ventricular contraction; for the percussion wave of Mahomed they substitute the term *papillary wave*, as they believe this to be due to the late and transient contraction of the papillary muscles; the tidal or predicrotic elevation is called the *outflow-remainder* wave, on the ground that it results from a continued contraction of the heart wall, which persists longer than that of papillary muscles.

Roy and Adami, in studying the pulse-curve, made use of an instrument called the sphygmometer (see *Practitioner*, vol. xlv. p. 29, 1890). With this instrument the authors claim that the pressure upon the artery can be regulated exactly; that the tracings are sufficiently large to enable one to distinguish minute variations in the characters of the pulse-wave; that the curves are free from inertia vibrations; and, lastly, that the blood pressure within the artery can be measured.

Views of von Frey and von Kries.—Von Kries and von Frey, in their interpretation of the pulse-curve, adopt the view that there is only a *single* heart wave, and that there are rebounds of this wave within the arterial system. Waves set up within an artery may rebound from the points of division of the vessel, but the chief site of rebound is the periphery of the vascular system—the capillary area. These waves modify each other by interference.

Von Frey concludes that the thorough discussion of any pulse-tracing is, strictly speaking, impossible in the absence of a knowledge of the number, origin, and direction of the waves which share in its production.

From recent observations by Leonard Hill and others it would seem not impossible that the degree of distension of the venæ comites in the radial sulcus may affect the pulse-tracing, as it may also mislead one in forming an estimate of the pulse by means of the finger. According to these observers, the radial sphygmogram must be interpreted as a pressure curve, for the movements of the lever of the sphygmograph are produced by the changes in pressure in the vessels which lie in the radial sulcus. We think undue importance has been ascribed to

venous influence, but we shall refer to the matter again under the section of pulse and respiration.

Digital Sphygmographs.—It must be borne in mind, however, that when the pulse is felt in the usual way, or when the pad of the sphygmograph is applied over its site, a much more

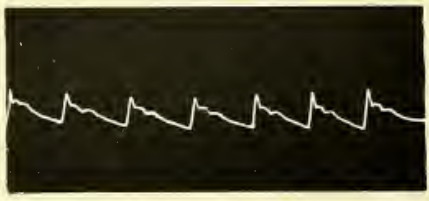


FIG. 18.—Tracing from the pulp of the middle finger (Dudgeon's sphygmograph) (identical with radial sphygmogram).

complex set of movements is obtained than is generally recognised. Thus the tracing is more than a diagram of the changes of blood pressure within the artery. It is perhaps more correctly to be regarded as a representation of a communicated pulsation from the vessel to the

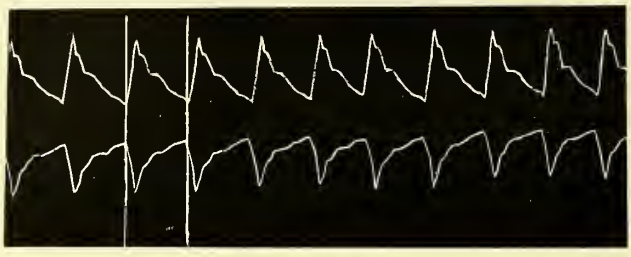


FIG. 19.—The upper tracing was taken from the radial artery in the usual manner; the lower one was taken with the pad of the sphygmograph lying alongside the artery. The lower tracing is an inverted sphygmogram.

tissues and liquids surrounding it. Thus the writer has taken tracings from the pulp of the middle finger by means of Dudgeon's sphygmograph, which exhibit all the features of an ordinary radial sphygmogram (*see* Fig. 18). Digital sphygmographs which record the pulsations of the vessels of the nail bed have been in use during the last few years, and Waller has recently described a very useful instrument of this kind under the name of the "onychograph."

The Inverted Sphygmogram.—It must not be forgotten also that there is some displacement of the whole artery in its bed at each systole of the ventricle. Allen Thomson believed this locomotion of the artery to be produced by the distension and elongation of the larger vessels near the heart, and in many persons the radial artery may be seen to move under the skin. These displacements are most easily seen in atheromatous arteries at their curvatures, and where they adhere to surrounding tissues. There is sometimes a considerable movement of the radial artery at the time of the ventricular systole, and there is a consequent "sucking-in" of the tissues adjacent to it. This is well shown in the tracing, Fig. 19, where the pad of the sphygmograph was placed alongside a large radial artery. The lower tracing in the figure shows that the needle of the instrument instead of being shot upwards at the time of systole, as

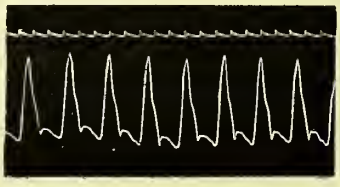


FIG. 20.—Systolic wave as a single summit—blood pressure relatively low.

in the upper tracing, is drawn backwards, thus forming an inverted diagram of the normal curve. Mackenzie figured similar tracings to show the effects of the sudden movement of the carotid artery in the neck.

General Information afforded by a Sphygmogram.—From a good sphygmogram we obtain information on many points in connection with the heart and circulation: (1) we ascertain the rate and rhythm of the heart; (2) we become acquainted with certain events occurring during the cardiac systole; (3) we get some indication of the state of the blood pressure; (4) we can form some idea of the state of the arterial walls.

We can only judge the height of the blood pressure in a general way from the form of the pulse-curve. When the systolic wave appears as a single summit, we may usually assume that the blood pressure is relatively low (Fig. 20). When a large systolic wave is divided by a notch, and the second summit is large and well sustained, the pressure in the vessels is generally high, but is not always so in people over sixty years of age (Fig. 21).

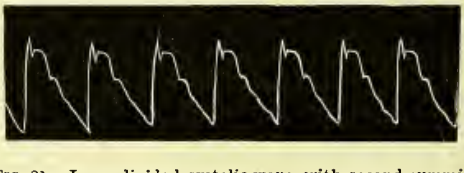


FIG. 21.—Large divided systolic wave, with second summit well sustained—blood pressure high.

Some Features of the Sphygmogram due to Instrumental Defect.—There are certain well-marked features in many tracings made with Dudgeon's sphygmograph which are undoubtedly due to a defect in the instrument. It is well to recognise this, and defects thus caused are not altogether undesirable so long as their undue exaggeration is avoided. With care it is

possible to do so, but in some low-pressure pulses the interpretation must always be somewhat uncertain. Thus when the needle is jerked up very forcibly the apex of the systolic wave is very pointed, or may even be looped (Fig. 22). The needle, from its momentum, has formed an apex to the curve which is out of all proportion. When this happens, the fall of the needle is also apt to be too extensive, and may come down to the level of the base line or below it. The lowering of the blood pressure during the diastole of the ventricle will thus appear to be very sudden and extreme, which it really is not (Fig. 23).



FIG. 22.—Looping of systolic wave (instrumental defect).

The Anacrotic Pulse and the Bisferiens Pulse.—These two forms of sphygmogram may be conveniently considered together, as a description of the former will include most of the features of the latter. The writer sees no reason to regard them as separate types.

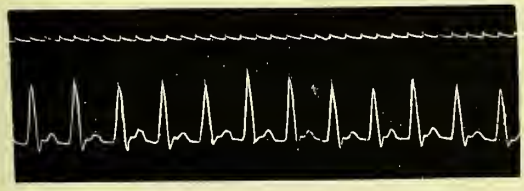


FIG. 23.—Too great falling of the first summit of the systolic wave (instrumental defect).

The *anacrotic* pulse may sometimes be a true representation of the course of events in the artery, and it is generally supposed to be characteristic of marked obstruction to the outflow of blood from the ventricle into the aorta, *e.g.* aortic stenosis. Graham Steell, in describing the pulse in aortic stenosis, recognises two distinct varieties: (1) the anacrotic pulse; and (2) the bisferiens pulse. According to this authority, the former is the more characteristic pulse in narrowing of the aortic orifice, owing to its sluggish development and infrequency; the latter has a more perpendicular up-stroke, a pointed percussion wave, and the cleft between this wave and the tidal wave is very deep. The tidal wave itself is pointed, and the aortic notch is usually situated low down in the line of descent. The dicrotic wave is usually, but not always, ill-developed.

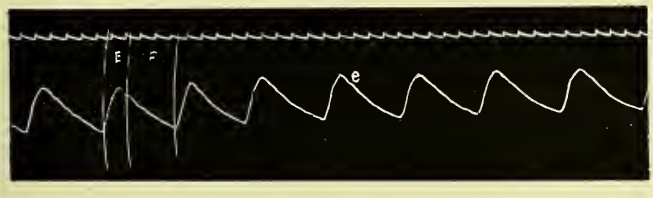


FIG. 24.—Anacrotic pulse, where the systolic wave appears as a rounded summit. E corresponds to the systole of the ventricle; F corresponds to the diastole of the ventricle. High position of dicrotic notch (*e*).

Steell thus describes the features of the



FIG. 25.—Typical anacrotic pulse from case of aortic stenosis.

anacrotic pulse:—(1) The up-stroke slopes instead of being perpendicular; (2) the percussion wave is practically abolished; (3) the tidal wave forms the summit of the curve; and (4) the dicrotic wave is usually ill-developed. In addition he notes the characteristic slowness

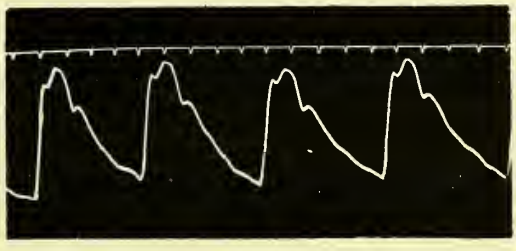


FIG. 26.—Pulse in aortic stenosis. (Von Jacquet's sphygmograph) (anacrotic type).

of the pulse, which is an important point. This description coincides with Mahomed's idea of the simplest pulsatile movement in an elastic tube, where the pulse-wave, being slowly delivered, is not modified by the percussion element. In the anacrotic pulse the systolic wave may in some instances appear as a single rounded summit (Fig. 24), or under other conditions there may be a distinct break in the line of the up-stroke (Figs. 25 and 26). Among other factors the delicacy of the sphygmograph is responsible for the degree of development of this notch. The ventricle, for one reason or another, has some difficulty in delivering its contents into the aorta, and hence there is not the usual sudden sharp impact imparted to the column of blood in the arterial system. The systolic wave then, if graphically recorded, will appear as a single sluggish elevation or as a divided summit, according to the delicacy of the instrument employed. The notch may occur at any part of the up-stroke, and sometimes it occurs at the top of the curve, thus giving rise to a double summit (Fig. 27). The second summit tends

to rise more and more above the level of the first-formed apex in the so-called anacrotic pulse, and is usually well sustained, but this last feature depends on other factors. If there is a relatively powerful left ventricle, and perhaps some pyrexia, the two waves forming the summit may be equally large and pointed,



FIG. 27.—Anacrotic pulse. Shows the notch dividing the two summits of the systolic wave at the top of the curve.

and the cleft between them may be very deep. The up-stroke is then more sudden and of greater amplitude. *Exaggeration* of these waves is of instrumental production, and it is in this way that the bisferiens pulse is formed (Fig. 28). The writer does not think this type of sphygmogram occurs in cases of pure aortic stenosis

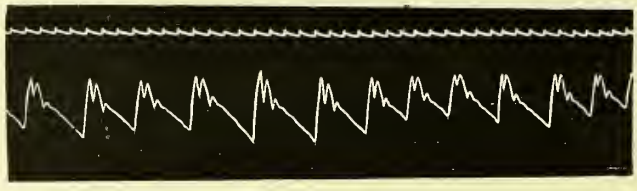


FIG. 28.—Pulse from a child suffering from tubercular meningitis which has assumed the *bisferiens* type under the influence of slight pyrexia. This pulse, when the vessels were more contracted, gave an *anacrotic* curve.

unless there is an elevation of the temperature, or at any rate some diminution in the peripheral resistance. A form of curve very similar to the pulsus bisferiens is commonly obtained in cases of aortic incompetence without stenosis, but the second summit is then rarely so high as the primary one (*see* Fig. 17). The double summit under these circumstances is entirely dependent upon the sudden and forcible manner in which the systolic wave is developed in the relatively empty arterial system.

The writer would define the *bisferiens* pulse as one in which the systolic wave is broken up into two more or less pointed summits of about equal size, the second summit *always* rising to a higher level than the first. This last feature is the important one, and it belongs also to the anacrotic pulse. Under certain circumstances the anacrotic pulse may become modified into the bisferiens pulse, and, generally speaking, the former coexists with a relatively higher blood pressure than the latter.

To return to the anacrotic pulse, we may state that obstruction to the outflow of blood at the junction of the ventricle and aorta is not the only, or even the chief, condition which

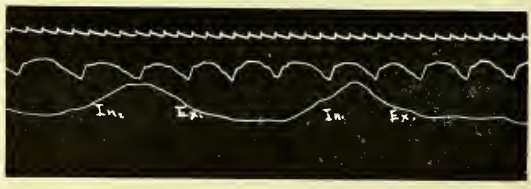


FIG. 29.—Tracing from a plexiform angioma of the scalp, showing the anacrotic character of the pulse-curve. The lower tracing is a respiratory one.

produces this modification of the systolic wave, though some of the most characteristic tracings may be obtained in aortic stenosis. Anything which tends to abolish the percussion element in the wave and which leads to the more gradual transmission of the impulse within the arterial tube, will produce a rounded or anacrotic curve. Tracings made with the plethysmograph of the pulsation of the arm or leg as a whole are generally anacrotic. The normal form of intracranial pulse, taken from the fontanelle or from a gap in the skull, is anacrotic.

The writer recently made tracings from a plexiform angioma which pulsated as a mass under the scalp of a child aged seven years. The type of curve obtained was also anacrotic (*see* Fig. 29).

In very young infants the sphygmogram of the radial artery is frequently anacrotic, and the same kind of pulse-curve is obtained in some cases of aneurysm of the aorta in adults, for reasons which will be stated hereafter. But in the opinion of the writer the most powerful

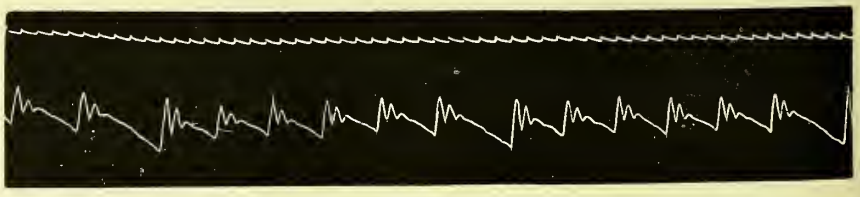


FIG. 30.—Pulse of child aged 5 years, suffering from tubercular meningitis. The peripheral arteries have become somewhat relaxed, and the usual anacrotic character of the curves has disappeared. The second summit of the systolic wave is still well developed. The type of pulse irregularity in this disease is well shown.

single factor in the production of the anacrotic pulse is *contraction of the peripheral vessels*. In children he is convinced that this is so, and in some cases of tubercular meningitis, where even the larger arteries (radials) are tightly contracted, this form of sphygmogram is very characteristic. Sometimes, when the child is

slightly feverish, the two elevations composing the summit may be of nearly equal size, and if the vaso-constriction be more completely removed, the second summit may be smaller than the first, but is still remarkably developed (Fig. 30). When the radial artery is felt to be contracted in this manner, the systolic wave is invariably rendered anacrotic, or is divided into a double summit; and should the vessel relax somewhat it is quite possible, as Graham Steell states, for the finger to appreciate the two summits of this wave. The writer has frequently known such a pulse called dicrotic. He can also confirm Steell's observation as to the development of this feature on one side of the body only; in carotid tracings it is not unusual to find that from the left side anacrotic, while that from the right side is normal—katacrotic.

A very good example of the anacrotic pulse is shown in Fig. 31. It was obtained from a boy fourteen years old, who had extreme hypertrophy of the left ventricle and remarkable thickening of the arteries all over the body. It was with great difficulty that the pulse could be felt in the radial, or even in the brachial

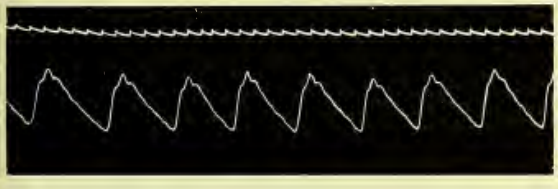


FIG. 31.—Very marked anacrotism of the pulse in a case where the peripheral resistance was enormously increased.

artery, and the presence of Raynaud's phenomena in the hands and feet suggested considerable obstruction in the peripheral blood-supply. The boy died from a cerebral hæmorrhage, the result, doubtless, of the excessively high arterial pressure. The whole arterial system was found to be uniformly thickened and contracted

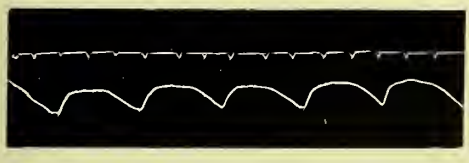


FIG. 32.—The fontanelle pulse taken by means of a system of tambours (rounded, slightly anacrotic curve).

almost to the obliteration of the lumen in some of the smaller branches. The left ventricle showed pure concentric hypertrophy, and the aorta was greatly narrowed throughout its whole course. Here the peripheral resistance must have been enormous, and this was probably the factor mainly concerned in modifying the systolic wave in the manner described. Compression of the abdominal aorta will alter the systolic wave in a similar way, for a normal pulse-curve can by this means be made to exhibit anacrotic characters.

The anacrotic sphygmogram is sometimes a true representation of the course of events in the artery, but it may also be obtained by faulty application of the sphygmograph. Such a curve must often, for this reason, be regarded with suspicion as being possibly an incorrect record of the pulse under examination.

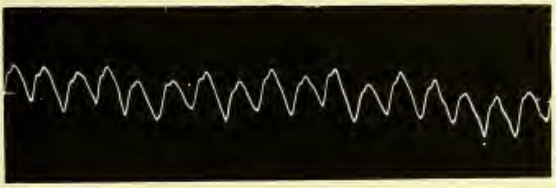


FIG. 33.—Fontanelle pulse from infant æt. 4 months, of typically anacrotic character. Normal form of intracranial pulse. (Dudgeon's sphygmograph.)

The Fontanelle Pulse.[1]—In the young child the brain, surrounded by fluid, is contained within the unyielding cranium. At one point there is a small area of elastic membrane—the fontanelle. The impulse of the left ventricle—systolic wave—is propagated to the arteries which ramify in the brain substance, and the whole organ expands. The fluid surrounding it is then compressed, and the membranous area is raised slightly. This is one of the fontanelle movements—the arterial—and it is really a plethysmographic pulsation. In this case the systolic wave is acting against great resistance, so the percussion element is largely abolished. This pulsation of the fontanelle is the expression of the more or less gradual swelling of the brain; and if a tracing be made by means of a system of tambours, a single slowly rising wave is often all that is obtained, or the curve may be slightly anacrotic (Fig. 32). But by means of more delicate apparatus, *e.g.* Dudgeon's or Marey's sphygmograph, the single wave can be broken up into a curve which is, as a rule, typically anacrotic. This is the normal form of intracranial pulse, and especially so when the general arterial blood pressure is high (Fig. 33).

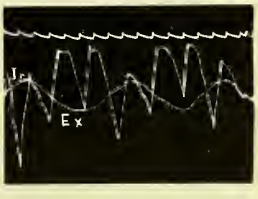


FIG. 34.—Simultaneous tracings of the respiratory movements and the fontanelle pulse to show the smaller arterial pulsations during inspiration, and the larger ones during expiration. (The intracranial tension was low.)

Another movement of the fontanelle occurs with respiration. With inspiration blood is sucked out of the venous sinuses of the brain towards the right heart,

[1] For fuller information on this subject the reader is referred to a valuable paper by Drs. Robert Hutchison and George Elder, *Ed. Hosp. Reports*, vol. iii. p. 268.

and the volume of the brain is thus diminished. Hence the membrane is drawn inwards, and the arterial pulsations which occur during the inspiratory phase are of smaller size (Fig. 34). Exactly the opposite thing happens during expiration—distension of the brain and bulging of the fontanelle. With a high intracranial tension—bulging fontanelle—the respiratory undulations become less marked, while with a low intracranial tension—depression of the fontanelle—they are well developed.

The Interpretation of Features due to Instrumental Defect.—Some of the features in the sphygmogram which we know not to be dependent upon true arterial waves may, nevertheless, be turned to good account. The *height* of the up-stroke made by the needle depends so much



FIG. 35.—Sphygmogram from radial artery during an attack of dyspnœa with marked contraction of the peripheral arteries. Radial calibre 2·4 mm. Systolic wave slanting, blood pressure relatively high.

upon conditions and methods of applying the instrument that it has, as a rule, little significance. But in some instances, where a series of tracings is carefully made on the same patient when there is reason to believe that his radial calibre is undergoing daily variations, then the height of the up-stroke is of some suggestive moment. It will afford some indication of the state of *tonus* of the vessel. In the tightly contracted state the play of the arterial wall will certainly be much less than when the artery is fully relaxed. The writer has taken many sphygmograms to illustrate this point, and has confirmed the results obtained by measurements of the calibre of the radial artery by means of Oliver's arteriometer (Figs. 35 and 36). Tracings made under the influence of amyl-nitrite

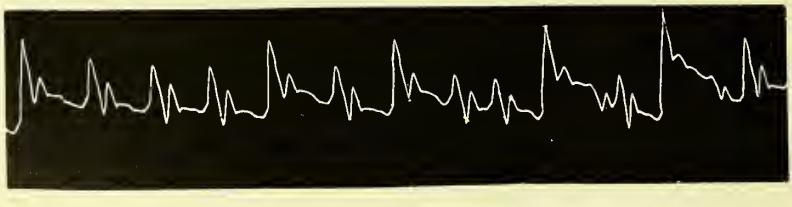


FIG. 36.—Tracing from the same case after treatment by nitrites, showing increased volume of pulse, larger and more vertical systolic wave; exaggerated dicrotism, especially in the abortive contractions of the ventricle. Arteries are relaxed—radial calibre 2·7 mm.

prove that the *height* of the up-stroke is sometimes a feature from which certain conclusions may be drawn.

The *direction* of the up-stroke is of much greater importance. It may rise vertically—at nearly a right angle to the moving smoked surface; it may rise in a more slanting direction, or it may sometimes be distinctly bent inwards during its ascent.

A sudden rise of the needle indicates that there is a relatively strong ventricle, and that the distribution of the blood through the arterial system is unimpeded. This is seen in some cases of aortic regurgitation, and the systolic wave in such a tracing may have a double summit, and that of the first may be looped. The modification of the systolic wave in such cases is purely of instrumental origin—it does not imply coexisting stenosis. A similar summit may be obtained in any case where pyrexia has excited the ventricle and dilated the peripheral vessels. A distinguishing feature in the sphygmogram of aortic incompetence is the small size of the dicrotic wave and its position at the lowest part of the tracing (Fig. 37).

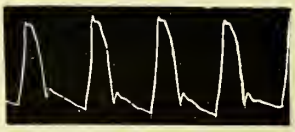


FIG. 37.—Pulse of aortic regurgitation. To show the small size of the dicrotic wave, and its position at the lowest part of the tracing.

When a relatively strong ventricle is acting against some peripheral resistance the up-stroke of the tracing may also be sudden, but a smaller systolic wave is produced. The summit is again divided, and the first apex may

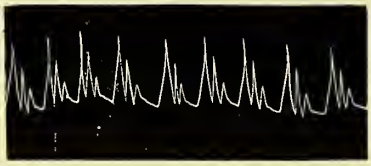


FIG. 38.—To show the kind of sphygmogram sometimes produced when a relatively strong ventricle is acting against considerable peripheral resistance. (Instrumental defect.)

be looped, acute, or rounded, while there is sometimes a considerable fall of the needle towards the abscissa before the second wave is developed. This second wave may be also unusually pointed owing to the inertia of the needle (*see* Figs. 30 and 38).

With a relatively weak ventricle and normal peripheral resistance, the up-stroke is apt to be more oblique; and if an instrumental apex to the systolic wave is formed, it is a small one. The up-stroke may be *incurved* where the ventricle is powerful, and the peripheral resistance increased. It is an instrumental defect (Fig. 39).

With regard to the fall of the needle it may

be said that a slow and even fall, with little prominence of the dicrotic wave, argues a relatively high blood pressure; a low pressure in the arteries is usually, but not always, distinguished by a rapid descent of the needle, and



Fig. 39.—To show the incurved up-stroke—an instrumental defect.

by a well-developed dicrotic wave. The excursion of the needle is generally greater when the blood pressure is low.

The Value of the Sphygmogram as indicating the State of the Blood Pressure

(*a*) *The Position of the Dicrotic Notch.*—The question of blood pressure is only partially discussed by the behaviour of the needle of the sphygmograph. In forming an estimate of the arterial pressure from a pulse tracing, we should specially note the *position of the dicrotic notch* on the down-stroke of the curve. *The higher the position of this notch the higher the blood pressure.* Its height above the base line of the tracing is therefore an important point in deciding this question. The tracing, Fig. 40, was taken from the case already described, where great hypertrophy of the left ventricle coexisted with general thickening and narrowing of the arteries (*see* p. 401). The height of the blood pressure in this instance must have been exceptional, and it is interesting to note the remarkably high position of the dicrotic notch above the base line of the tracing. The low

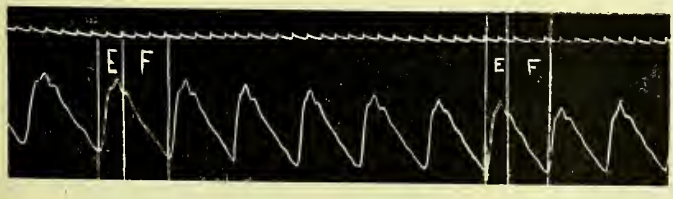


Fig. 40.—To show the high position of the dicrotic notch above the base line of the tracing. Indicates a high arterial blood pressure. E, systole of ventricle; F, diastole of ventricle.

position of the notch in aortic regurgitation may be usefully compared with this (*see* Fig. 37).

(*b*) *The Character of the Dicrotic Wave.*—This is also suggestive. If the dicrotic wave has been absent or very small in a sphygmogram of the low-pressure type, and gradually becomes more developed, we may safely affirm that the arterial pressure is increasing. Conversely the gradual disappearance of the dicrotic wave in a case of this kind denotes that the blood pressure is falling to a dangerously low point. From a tracing of this type, if it persist, one may most always prognosticate a fatal issue, and in the pneumonias of young children the writer has not so far found a single exception to this. A tracing is appended to illustrate this type of pulse from a child dying of broncho-pneumonia (Fig. 41).

Should an increase in the blood pressure occur in a case of this kind, the first indication is the appearance of a dicrotic wave. Therefore this can always be regarded as a reassuring sign. In the rapidly dilating heart of pneumonia and acute rheumatism in children, it has been elsewhere pointed out that the most significant and striking symptom is often the disappearance or great diminution of the dicrotic wave. A case of extreme rheumatic dilatation in a young child showed this feature when first examined, and there was also epigastric pulsation. After a few days' rest in bed, recovery was indicated by the reappearance



Fig. 41.—Pulse and respiration in a case of fatal broncho-pneumonia. Child æt. 1¾ years. To show the disappearance of the dicrotic wave—blood pressure dangerously low.

of the dicrotic wave in the pulse-curve, and by the disappearance of epigastric pulsation.

(*c*) *The Significance of the Dicrotic Wave.*—The presence of the dicrotic wave indicates that there is some degree of arterial pressure, for the pressure must be excessively low when the recoil wave is not sufficiently powerful to be reflected from the aortic valve. The summit of the dicrotic wave is usually less pointed than the systolic wave.

Dicrotism and Hyperdicrotism.—In the so-called dicrotic pulse the dicrotic notch bears a certain relationship to the base line of the trace. If it descend to the base line before the dicrotic wave is developed, the pulse is sometimes called *fully dicrotic*. Should the notch occur below the level of the abscissa the pulse is said to be *hyperdicrotic*. This may occur to such a degree that the dicrotic wave runs into the up-stroke of the next beat. The *rate* of the pulse, combined with increased inertia of the needle, plays a very large part in the production of this exaggerated dicrotism. Hyperdicrotism is always associated with great frequency of the pulse,

and with general relaxation of the vessel (Figs. 42, 43, and 44).

The Monocrotic Pulse.—It is somewhat difficult to decide upon the form of pulse-curve to which this term may be correctly applied. The usual definition of a monocrotic pulse is one in which the dicrotic wave is absent. The sphygmogram shows a series of single curves, but in some of these the position of the dicrotic notch is indicated either by a break towards the end of the down-stroke, or by a slight rise which appears just before the commencement of the up-stroke (Fig. 45). A monocrotic pulse, in its literal sense, means a pulse which shows only one distinguishable wave. But there are certain fallacies to be noted.

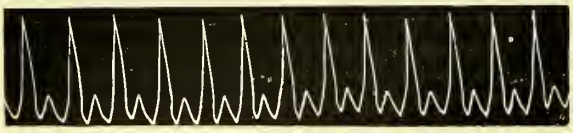


FIG. 42.—Typical dicrotic pulse (enteric fever, 20th day).



FIG. 43.—More fully developed dicrotism.

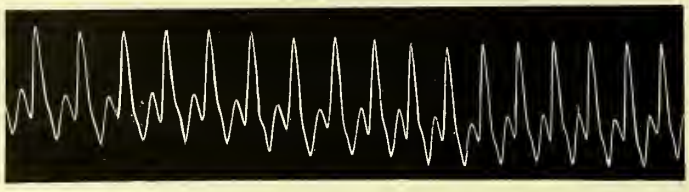


FIG. 44.—Hyperdicrotism of the pulse. This is largely the result of increased inertia of the needle of the sphygmograph and extreme rapidity of the pulse.

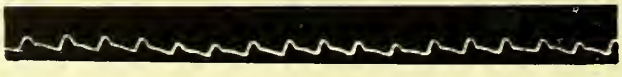


FIG. 45.—*Monocrotic* pulse (disappearance of dicrotic wave) in fatal case of puerperal septicæmia.

In the case of very young infants, and also in older children, the sphygmogram sometimes appears as a single undulation (Fig. 46). Hence the pulse at this period of life has been called monocrotic, and this would seem to be compatible with the low blood pressure of the infant (Ozanam, Keating and Edwards, and others). By the use of more delicate instruments, however, the infant's pulse is found, in reality, to be of the type which is associated with a *relatively* high blood pressure in the arteries.

Again, when the arterial pressure is low and the pulse rapid, the dicrotic wave may be developed to such an extent that it nearly equals the size of the systolic wave. Such a tracing as Fig. 47, where the diastolic phase of the ventricle is hardly apparent, might be mistaken for a monocrotic pulse.

Speaking of the monocrotic pulse, Mahomed said: "A monocrotic pulse is merely a hyperdicrotic pulse, which is so rapid that the primary up-stroke becomes superimposed upon the dicrotic wave, which has not time to travel to the radial before the more rapidly transmitted primary wave of the succeeding cardiac contraction overtakes and absorbs it, as one often sees a small wave of the sea merged into the larger one which has overrun it.

In a very rapid hyperdicrotic pulse we have observed this for a few beats at a time (*see* Fig. 45), but in such a case the *dicrotic wave* is *not* really *absent*, nor is the blood pressure excessively low; the mere rate of the pulse has led to the production of monocrotic curves. It might be advisable to limit the term "monocrotic pulse" to those cases in which the single elevation is the result of *the disappearance of the dicrotic wave.* A sphygmogram of this type is of evil omen, because it denotes extreme lowering of the blood pressure; the hyperdicrotic form, which becomes monocrotic, is only significant of danger on account of its great rapidity.

The Sphymographic Appearances of the Pulse in Infancy

The radial artery of the new-born infant is very small, and cannot be felt as a definite structure. The vessel is situated rather deeply in the tissues of the wrist, and there is often a large quantity of subcutaneous fat around it.

Ozanam, Keating and Edwards, and others have described the sphygmogram from the new-born infant as a meaningless wavy line (Fig. 49). From the absence of dicrotism they conclude that the arterial pressure is very low, and regard the pulse as monocrotic. These authors



FIG. 46.—Pulse of infant aged 10 months, showing one wave (erroneously called *monocrotic*).

hold that the conditions necessary for the production of the dicrotic wave are absent in early

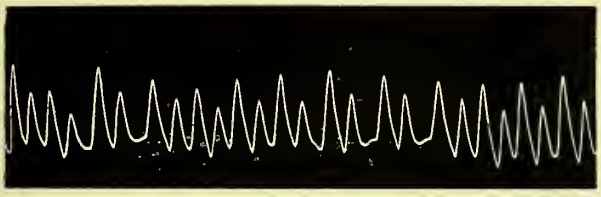


FIG. 47.—Another form of pseudo-monocrotic pulse. The dicrotic wave is greatly exaggerated.

life. These conditions, they say, are elasticity of the great vessels combined with low tension

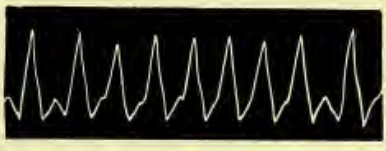


FIG. 48.—Hyperdicrotic pulse becoming monocrotic from increased rapidity.

of the peripheral circulation, and a sharp contraction of the heart. In the infant they



FIG. 49.—Pulse of the new-born infant (Ozanam). (Incorrectly called *monocrotic*.)

believe that, owing to the relatively less strong heart, the arteries are not distended to such an

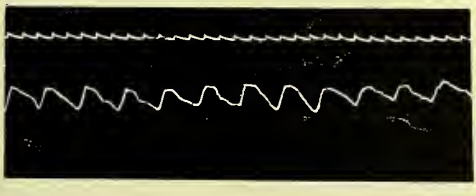


FIG. 50.—Pulse of an infant after ligature of the umbilical cord. (Indicates a relatively high blood pressure.)

extent as to cause a thorough recoil, and, further, that there is a marked resistance to the peripheral circulation in the kidney.

The writer's tracings from the pulse of infants do not show an absence of dicrotism, and the pulse-curve does not indicate a low arterial pressure. Sphygmograms made in the manner already described show clearly that the pulse of young infants is of the type associated with a *relatively* high blood pressure (Figs. 50, 52, and 53). Dicrotism is as marked in the sphygmogram of the new-born infant as it is in any high-pressure pulse.

A tracing was obtained from the posterior tibial artery of a fœtus during the process of "turning," and in it the position of the dicrotic notch is clearly indicated (Fig. 51). Even before birth, then, the pulse-curve would seem to be marked off in this way into systolic and diastolic portions, when we employ suitable means of instrumental examination.

The sphygmogram shown in Fig. 52, which was taken from a healthy boy six days old, brings out clearly all the features which characterise a high-pressure pulse-tracing in an adult. The systolic wave rises in a slanting direction, and the dicrotic notch occupies a high position on the down-stroke. The instrumental division of the systolic wave into two summits is just apparent in some of the curves, but in the tracing, Fig. 53, from an older child it is very well marked.

To find the explanation of this high-pressure type of curve, which is characteristic of the very young infant, attention may be directed to the striking resemblance which exists between it and that obtained in aortic stenosis in the adult. The tracing from a premature infant, and some which I have taken from babies a few hours after birth (Fig. 54), compare equally

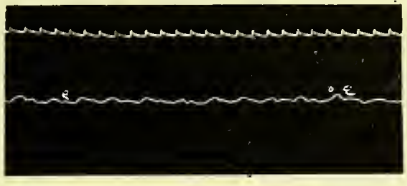


FIG. 51.—Tracing from the posterior tibial artery of a fœtus (Shows the dicrotic notch *e*.)

well with those from a case of aortic stenosis, and perhaps still better with the type obtained from some cases of aneurysm affecting the arch of the aorta. The analogy to the latter is a specially apt one, because the conditions giving rise to this form of pulse-wave in both instances are remarkably similar. We refer to the relation which exists between the size of the heart and the width of the aorta, and also to the fact that there is relatively less blood in the arterial system, and hence a smaller pulse at the wrist. Beneke has shown that the volume of the in-



FIG. 52.—Pulse of a normal infant 6 days old. (Indicates a relatively high arterial blood pressure.)

fant's heart is to the width of its ascending aorta as 25 : 50, whereas before puberty it becomes as 140 : 50. There is thus in both con-

ditions a left ventricle connected by a narrow opening with a relatively capacious aorta. On this account the ventricular contraction is slowly developed, and the "shock" imparted to the column of blood at the root of the aorta is more slowly propagated throughout the arterial system than it is under normal conditions. The "systolic wave" is thus modified in much



FIG. 53.—Pulse of a child aged 4 months. To show the division of the systolic wave into two summits.

the same manner as it is in the fontanelle pulse, and rises in a sloping direction instead of being a vertical line. The features of a high-pressure pulse are thus delineated in the tracing from a new-born infant for the same reasons that they are reproduced in aortic stenosis or aneurysm in the adult.

Until the end of the first year of life the



FIG. 54.—Pulse of a premature infant (7 months' gestation) when 10 days old. (Compare with pulse of aortic stenosis or aneurysm.)

infant's pulse remains as this high-pressure type, but after a few months the systolic wave is more distinctly divided into a double summit, and the volume of the pulse increases.

It is interesting to note the effects of pyrexia on the pulse of infants under a year old; a high degree of fever at this age does not render the pulse dicrotic or hyperdicrotic (Fig. 55). In adults the form of pulse-curve is very readily altered by even slight elevations of temperature, and varying degrees of dicrotism are produced. In children under one year old it is most unusual to obtain a pulse-tracing which shows a well-marked dicrotic wave, either as a result of pyrexia or from any other cause. At this age a dicrotic pulse is occasionally found in broncho-pneumonia, where the blood pressure is extremely low.

PULSE IRREGULARITY

Introduction.—Perhaps nothing strikes one so much in the study of the pulse as the frequent occurrence of irregularity. Children will sometimes exhibit a remarkable form of pulse, which it is difficult to reconcile with perfect health, and yet there is often no indication of disease. The peculiarity of the pulse-rhythm may be of a very transient nature, or it may be present over a considerable period. It may be noticed in the infant at birth, or soon afterwards, when the pulse-rate is unusually slow; this kind of irregularity disappears when the pulse becomes rapid.

The pulse-wave increases in strength and volume from birth to puberty, and the increase in size of the radial artery is proportionately greater than the growth of the hand. The *rate* of the pulse of the young infant, if carefully observed, will be found to vary enormously. When a child is kept in bed for a considerable period the pulse invariably slows down in a striking manner, and the same thing is noticed after a febrile attack. Under the influence of a high temperature the pulse will be very greatly accelerated, but after the fever subsides the rate becomes abnormally slow, and, at the same time, there is a tendency to irregularity of rhythm. During sleep the same type of slow, irregular pulse is often found. After the third year of life there is a notable diminution in the rate of the pulse. Great frequency of the pulse in young children has much less significance than in adults. The enormous rapidity at the commencement of a scarlatinal attack is sometimes striking, and in the later stages of tubercular meningitis a peculiar irregularity in a very rapid pulse is worthy of note—the number of beats counted in separate quarter minutes is found to vary remarkably.

Persistent slowness of the pulse in early life is unusual, and may suggest cerebral disease. But in ill-nourished, marasmic babies great slowing of the pulse is sometimes observed, and this symptom has been noted in cases of sclerema neonatorum. Catarrhal jaundice in children under six or seven years of age does not appear to

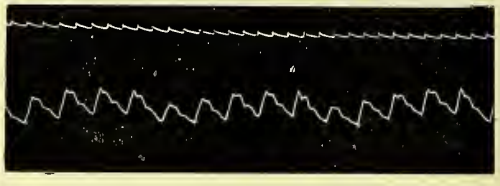


FIG. 55.—Pulse of a child æt. 11 months; temp. 105°. (Dicrotism has not been produced.)

produce a slowing of the pulse-rate, at least not to such a marked degree as is observed in adults.

The *rhythm* and *character* of the pulse are frequently of very great value. In the acute pneumonias of young children, if sphygmographic records be made throughout the course of the disease, important conclusions may be formed as to the favourable or unfavourable progress of the case, and of the good or bad effects of treatment.

A slow, irregular pulse is specially characteristic of cerebral tumour, abscess, or hæmorrhage. In children under two years of age suffering from tubercular meningitis, the writer believes the peculiar retardation of the pulse in the early stages of the disease is rarely observed. When it is present it is probably a very transient phenomenon. So far as his experience goes in these cases, the pulse at this age is generally very rapid from the time the child first comes under examination. But in tubercular meningitis in older children there is sometimes a type of irregularity which may be of some diagnostic value.

Classification of Pulse Irregularities.—Pulse irregularities have been classified in various ways, none of which are altogether satisfactory. There has been much misconception of the symptom, because writers have drawn distinctions between the terms "intermittency" and "irregularity" in connection with the subject. Such a distinction is in reality a purely artificial one, as will be afterwards shown. A pulse can only be called "*intermittent*," in the true sense of the word, when a whole heart-beat is, from time to time, entirely missed out, that is to say, the ventricle remains in a state of diastole for *exactly two ordinary pulse intervals*, and makes no attempt to contract during that time either prematurely or otherwise. This condition of matters certainly does not seem to occur in children, so we may dispense with the phrase "intermittent pulse" in connection with the pulse irregularities observed during early life. Cushny, in an interesting paper on intermittent pulse, has attempted to classify the common types of irregularity which occur in adults on the basis of cardiac physiology. In animals, by stimulation of the auricle and ventricle respectively, he has been able to produce and distinguish two main forms of pulse irregularity or intermission. The first is due to ventricular failure, and the second to auricular.

It will be necessary to shortly summarise his conclusions in order to be in a position to deal adequately with the subject of pulse irregularity.

The classification suggested by Cushny is worked out by observing what occurs in the behaviour of both the auricle and the ventricle during the so-called intermission in the arterial pulse. Now, clinically, it is only in exceptional cases that we have the opportunity of recording the auricular contractions. Dr. James Mackenzie has studied this subject in a most thorough and admirable manner, and by recording the pulsation of the jugular vein simultaneously with the radial pulse he has shown clearly what the right auricle is doing during the irregularity of the left ventricle. The left auricle, he concludes, in all probability acts similarly to the right one. The behaviour of the right ventricle he has also determined, in many cases, from graphic records of the pulsation of the liver or jugular veins, and he is led to believe that hemisystole of the heart does not occur. According to his numerous and highly reliable observations, the right ventricle does not act independently of the left, or very rarely so. Mackenzie has also shown that the auricles may retain their regularity without the ventricles necessarily following suit. That is to say, the ventricles may for some reason contract *prematurely* before the normal period of their diastole is finished, and the next auricular contraction will still come in at its proper interval after the preceding one. Under such circumstances the ventricle will not respond to this auricular contraction, but will wait for the next one.

In the majority of cases where we find pulse irregularity, we have to determine its nature by an examination of the *left ventricle* alone. It is seldom that the behaviour of the other chambers of the heart can be observed unless the case be one of advanced cardiac disease. Therefore it will be well, clinically, to classify pulse irregularities according to what is revealed by a combined examination of the arterial pulse and the apex beat. The cardiograph must in some cases be used to make a simultaneous record of the left ventricular movement, or, in some instances, the simpler method of listening to the cardiac sounds may be adopted.



FIG. 56.—The "intermission" is equal to two pulse-intervals (Cushny's classification).

Cushny's Classification of Pulse Irregularities.—Cushny recognises two *main* forms of pulse-intermission—

(1) Those in which the intermission is about equal to two pulse-intervals (Fig. 56).

(2) Those in which the intermission is shorter than two pulse-intervals (Fig. 57).

He describes *two varieties* of intermission belonging to each of these classes, and his classification is as follows:—

I. *True ventricular intermissions*, in which the pause is exactly equal to two pulse-intervals, and during which there is no cardiac sound.

In the human subject this is certainly a very uncommon form, and very great discrimination would sometimes have to be exercised in deciding whether such a tracing should not properly be relegated to the next division. The supposed "dropped" beat may not really occur, for the premature contraction of the ventricle

may be too feeble to show an elevation in the sphygmogram. The following is Cushny's description of what happens in animals:—

The auricular contractions occur at regular intervals, and the ventricles usually respond. But occasionally the impulse emitted from the

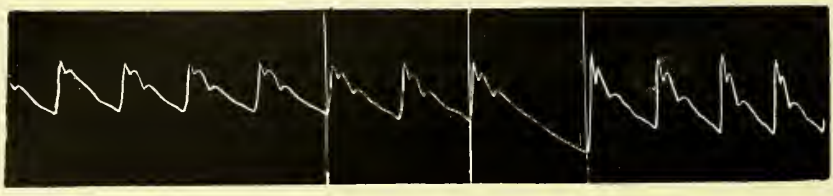


FIG. 57.—The "intermission" is *less* than two pulse-intervals (Cushny's classification).

auricle in regular course is not followed by a ventricular contraction. For some reason the ventricle does *not* contract, and thus a whole heart-beat is entirely missed out. The ventricle contracts in response to the next auricular impulse, but the *cause* of the intermission in this case lies with the *ventricle* itself, and not with the auricle, which sent down its impulse as usual. When this occurs, then, we may correctly describe it as a "true ventricular intermission."

The writer has only seen one tracing where this form of pulse irregularity could actually be shown to occur. He obtained it from Dr. Mackenzie of Burnley, who regards it as an almost unique example of the so-called *true* ventricular intermission (Fig. 58). Mackenzie believes that in the vast majority of intermittent pulses in adults, the ventricle really makes a second attempt to contract during the long pause, but that the effect

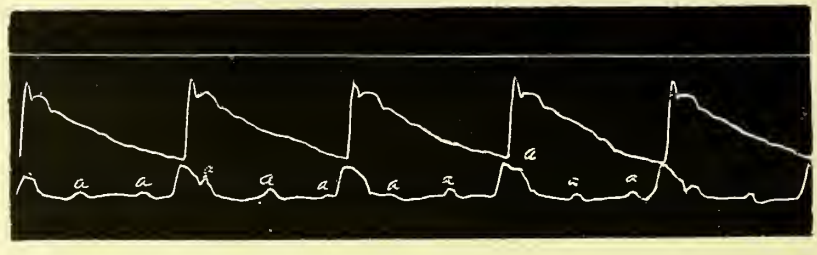


FIG. 58.—"True ventricular intermission." The lower tracing is from the jugular vein, and gives the auricular movements (*a* = auricular systole). (Cushny's classification.)

on the circulation is not made manifest at the radial artery. In the true intermission referred to, it is seen that the ventricle did not always respond to the auricular stimulation. A simultaneous tracing obtained from the internal jugular vein recorded the auricular movements. The venous tracing is somewhat difficult to interpret, but there is no doubt that the auricle contracts *twice* or *thrice* for each ventricular beat.

II. *False ventricular intermissions*, in which the pause is equal to two pulse-intervals, but it is often interrupted by a slight elevation.

In every case a first heart sound is to be heard during the intermission.

In this form, as in that just described, the auricle still contracts at regular intervals, and often the ventricle follows suit in the proper way. But sometimes the ventricle is unduly irritable, and is stimulated to contract from other causes than an auricular impulse. Therefore, after having given its usual contraction in response to the auricle, it goes off a second time during the period in which it should be resting and preparing to receive the next stimulus from the auricle. In doing so it exhausts itself, and is unable to



FIG. 59.—"False ventricular intermission." The "intermission" is equal to two pulse-intervals, but an elevation (*premature systole*) occurs during the pause. (Cushny's classification.)

contract when the auricular impulse descends. The diastolic period is therefore prolonged until the next auricular beat occurs (Figs. 59 and 60.)

III. *False auricular intermissions*, in which the pause is shorter than two pulse-intervals.

There is a slight elevation in the tracing during the intermission, and the stethoscope reveals a first heart sound very soon after the last regular pulse elevation.

This type of irregular pulse is very similar to the false ventricular intermission, but according to Cushny measurement in this case shows that the full beat and the premature beat are of *shorter* duration than two ordinary pulse-intervals. By this means he would diagnose the occurrence of this variety in man. It is probable that the false ventricular intermission is commonly mistaken for the auricular form, and measurement would not seem to be a reliable method of distinguishing the one variety from the other. The only *certain* method, as Mackenzie points out, is to obtain a record of

the auricular movement from the jugular pulse. This is only possible in the few instances in which venous pulsation is coincidently present, so as a general rule the "false auricular intermission" cannot be definitely diagnosed. In this variety the auricle contracts prematurely, and the ventricle follows suit; the regular contractions of the auricle are thus broken—hence the *cause* of the irregularity lies in the *auricle* and not in the ventricle.

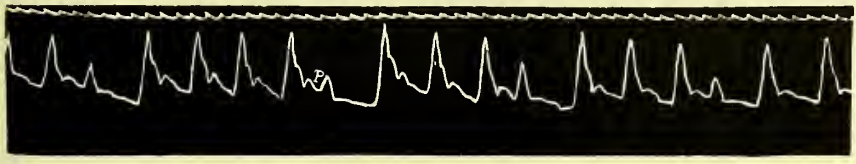


FIG. 60.—"False ventricular intermission." The ventricle has made a second and premature contraction (*p*) after its full beat. (Cushny's classification.) The last intermission shown in the tracing may be of the "false *auricular*" type. (*See* p. 408.)

IV. *True auricular intermissions*, in which the pause is shorter than two pulse-intervals, and during which there is no cardiac sound (Figs. 61 and 62).

In this variety, unlike the last described, the auricle instead of going off into a premature contraction at various intervals, does not contract at all. For some reason the sequence of regular auricular impulses to the ventricle is broken, and after a longer pause than usual the next contraction comes and is followed by the ventricular systole. Cushny calls this a true auricular intermission, but a study of the child's pulse shows it to be of such common occurrence in early life that another explanation is more likely. This kind of intermission is found in adults, but it only occurs at very rare intervals (*see* Fig. 57), whereas in the case of the child it occurs all through the tracing. The writer may also state that in adults who exhibited this peculiar irregularity, he could never make out other heart sounds after the regular contraction; the ventricle may, nevertheless, have made an attempt at premature contraction. Still there is no reason why, in both adults and children, this form of pulse should be regarded as an intermission at all. There is no real *intermission* of the heart—that is, as a beat *lost*; it may be more simply and more correctly described as a beat *delayed*.



FIG. 61.—"True auricular intermission." The "intermission" is *shorter* than two pulse-intervals, and there is no second contraction of the ventricle during the pause. (Cushny's classification.)

These four types of pulse intermission described by Cushny may be obtained experimentally on animals, but, clinically, if we adopt his nomenclature, there are only two common variations which we need consider, namely, the so-called *false ventricular intermission*, and the *true auricular* one. The true auricular intermission of Cushny is the one form of irregularity which is very frequently found in children.

Mackenzie, who has investigated the subject of pulse irregularity from the clinical side in an exhaustive manner, adopts a much simpler and more useful classification. He admits the two common types which have been mentioned; the so-called false ventricular and the true auricular intermissions. The two other forms of irregularity described by Cushny apparently do exist, but one has not often the opportunity of confirming the auricular contraction in cases of doubt.



FIG. 62.—Pulse of child æt. 5 years, showing irregularity of the "true auricular" type, according to Cushny's classification.

Mackenzie's Classification of Pulse Irregularities.—Mackenzie, in his classification of irregularities of the pulse, suggests two groups—

I. Those in which the duration of the period of ventricular *systole is regular*. The irregularity is here shown by the varying lengths of the diastolic periods (Fig. 63). This is the type observed so often in the young, and is what Mackenzie terms "the youthful type of irregularity." It occurs in adults too, however, and is evidently the same thing as Cushny has described as a true auricular intermission.

II. Those in which the duration of the period of ventricular *systole is variable*. This is the usual type in adults, and has been supposed never to exist in young children. The writer has, however, found it on several occasions, the youngest child being only two years old

(Fig. 64). It may occur also in older children who suffer from advanced cardiac disease. This is the same form of irregularity as Cushny describes under the head of false ventricular intermission (Fig. 65).

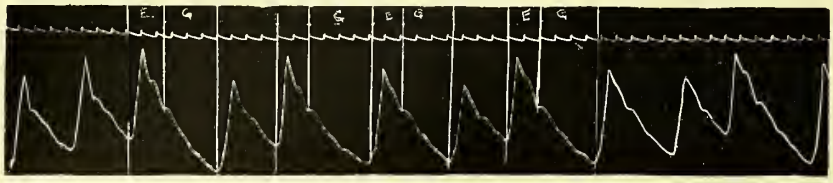


FIG. 63.—Pulse of child æt. 4½ years. E, The duration of the period of ventricular *systole* is *regular*; G, the duration of the period of ventricular *diastole* is *variable*. "*Youthful type of irregularity*" (Mackenzie's classification).

According to Mackenzie's classification, then, we find that practically all the pulse irregularities met with in children fall into the first group, while the commonest type of adult irregularity belongs to the second group.

Such a method of classification has done much to simplify the study of this interesting subject, but there are many kinds of pulse irregularity in children which have hitherto received no attention. Respiration is a most important factor in the production of some of these.

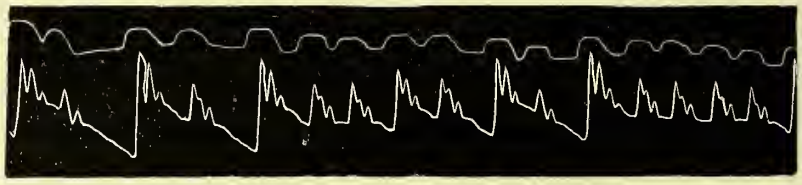


FIG. 65.—The duration of the period of ventricular *systole* is *variable*—the upper tracing is a cardiographic record. "Adult type of irregularity" (Mackenzie's classification). This is the "false ventricular intermission" of Cushny.

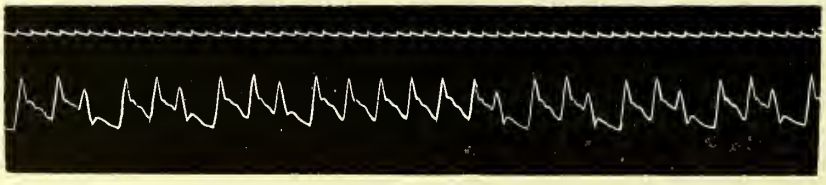


FIG. 64.—"Adult type of irregularity" (false ventricular intermission) occurring in a child æt. 2 years during the course of a mild croupous pneumonia. (Mackenzie's classification.)

The Characteristic Type of Pulse Irregularity in the Child.—All children at some time or other exhibit a form of pulse irregularity. When a tracing is taken the precise nature of the irregularity is well shown, and it is found that the systolic portions of the pulse-curves are regular in their duration. If we measure with a pair of compasses from the middle of the up-stroke to the dicrotic notch, we find the length about the same in all the curves. If we measure in a similar manner from the dicrotic notch to the commencement of another up-stroke, we find the distances to vary considerably. In the child's pulse, then, as Mackenzie first pointed out, the *equal duration of the systolic periods* is much the most constant feature, while very frequently *the diastolic periods are of unequal length* (Figs. 66 and 67).

In the very rapid pulse the duration of the ventricular systole is not much shortened; it is at the expense of the diastolic period that things are balanced. Thus we can understand that when a child's pulse is very fast the diastolic phases tend to shorten, and hence this type of irregularity disappears more or less entirely. In a tracing of a very rapid pulse one can easily observe the relatively greater duration of the systolic portions of the curves, and the diastolic portions are reduced to a minimum. The converse of this is true, for when the rapid pulse begins to slow down again we generally find undue prolongation of the diastolic periods, and there is no very marked change in the duration of the systolic periods.

The pulse generally becomes slower during sleep, and then the occurrence of this kind of irregularity can often be noted. When the child wakes the pulse tends to become faster, and thus becomes more regular. The pulse of the new-born baby is, as a rule, very rapid, and irregularity of the kind mentioned is not usually



FIG. 66.—The characteristic type of pulse irregularity in a child æt. 6 years, with simultaneous tracing of respiration. Duration of systoles (E) are regular. Duration of diastoles (G) are variable. It may be noted that the irregularity is here independent of respiratory causes (*see* p. 411).

seen. But should the pulse become temporarily slower the prolonged diastolic phases will soon make their appearance. This is a common type of pulse irregularity in animals (Fig. 68).

As the child grows older there is a *natural slowing* of the pulse. It is probable that the physiological inhibition of the heart is not fully developed in early infancy, hence the great frequency of the beats during the first few months of life. It has been found that stimulation of the vagus has not the same restraining influence upon the heart-beats in infants that it has in adults. Therefore we may find the explanation of this peculiar pulse irregularity,



FIG. 67.—The characteristic type of irregularity in a child æt. 4 years. Systolic periods (E) equal; diastolic periods (G) unequal. The shorter pulse-beats would seem to occur during the *inspiratory* part of respiration (*see infra*).

which is certainly most strikingly developed at the time when the pulse is naturally slowing down after infancy, in the fact that the vagus is beginning to exert more decided control over the heart.

The Causes of Pulse Irregularity in Children.—This type of pulse irregularity in its most marked form would certainly appear to be a purely nervous phenomenon. Cushny calls it a "true auricular intermission." He says the emission of the impulse from the auricle to the ventricle is, for some reason, *retarded.* The ventricular rhythm is dominated by the auricular, so the ventricle must wait till the impulse descends to it. There is no reason, he points out, why the auricular contraction should be retarded for a whole cycle, and, as a matter of fact, it almost always occurs shortly after its proper time. This is quite an exact description, but the writer does not consider that it is either necessary or advisable to speak of this frequent *halt* in the pulse as an *intermission.* A whole heart-beat is not missed out, and he cannot agree with Cushny that the *auricle* is the controlling influence in the production of the irregularity. In animals it may be so, and perhaps also in adults, where an intermission which might be of this nature occurs at very rare intervals, during which no cardiac sounds can be heard.

In children, at all ages, the writer has made very numerous observations on this type of pulse irregularity, and the matter has been studied from various points of view.

The Influence of Respiration on the Pulse-Rhythm.—By taking simultaneous records of the pulse and respiratory curves, it has been possible to note the effects of inspiration and expiration on the form and duration of the pulse-wave. The movements of inspiration and expiration in the child apparently influence the radial pulse in a more marked manner than is generally appreciated, and to a greater degree than is usually observed in adults. By deep respiratory movements there are great alterations produced in the quantity of blood reaching the medulla. Diminished or increased blood pressure in the vessels of the medulla will lead to depression or stimulation of the various centres resident there, and thence to the development of certain effects on the heart and blood-vessels. We do not mean to assert that the youthful type of pulse irregularity is *always* or even *usually* due to the influence of respiration on the vagus centre, but in many cases the pulse tracing may show a distinct respiratory rhythm of this nature (*see* Fig. 67). We would go the length of saying that respiration, either directly or indirectly, is intimately associated with the production of the so-called youthful type of irregularity. In some instances, however, it is difficult to see how it participates, for when a child holds its breath the irregularity is still as marked as ever, and in other cases one finds both the usual form of irregularity and also another, which is clearly due to respiration.

The Influence of the Vagus.—This youthful type of pulse irregularity is by no means uncommon in young adults of nervous temperament. Perhaps the pulse, when first examined, becomes unduly rapid, and the vagus control is



FIG. 68.—Sphygmogram taken from the leg of a young dog to show the "youthful type of irregularity"; is probably due to respiration.

temporarily overbalanced. As the vagus begins to regain its influence a slowing of the pulse occurs, and then its halting, hesitating character becomes apparent. We therefore believe that this form of irregularity is largely the result of *over-action on the part of the vagus.* Such over-action may, of course, be dependent on many causes, among which respiration has already been mentioned as one of the most constant and important. An over-acting vagus produces the retardation noticed in some of the pulse-beats, or, in other words, results in the occasional *delay* of a systolic contraction (Fig. 69). Under the action of digitalis a pulse irregularity of this kind is often produced (Fig. 70).

In the youthful type of irregularity the diastolic period is sometimes so much prolonged that the whole pulse-curve, if measured, may be found equal to, or even greater than, two ordinary pulse intervals. But a second heart-beat does not really occur in this form of irregularity.



FIG. 69.—The longer pulse-beats (lengthened diastolic periods) occur during expiration. The vagus is here regaining its influence over the heart (*over-action*), after the transient loss of control at inspiration (quickening of the pulse).

Some Conditions where this Type of Pulse Irregularity occurs.—The conditions favouring the appearance of irregularity in children are:—

(1) Rest in bed.

(2) Slowing of the pulse after fevers.

(3) During certain diseases—chorea, tubercular meningitis, etc.

(4) During respiration.

Possible Subdivision of the Youthful Type of Pulse Irregularity.—The irregularity in all these cases is very similar, but the writer is disposed to think that certain distinctive features mark that form which is dependent on respiration. In the usual type of irregularity, which is *not* due to respiration, the diastolic periods are sometimes lengthened, and are sometimes markedly shortened. Short beats follow long beats all through the tracing without maintaining any definite order. In the respiratory irregularity the diastolic periods are almost invariably lengthened during the expiratory phase, and are sometimes shortened during inspiration. When such a tracing is examined a more regular type of irregularity is apparent—there is some method in the arrangement of the long and short beats. It is therefore possible to make a *subdivision of the youthful type* of pulse irregularity:

(1) Those which are due to the influence of respiration.

(2) Those which are *not* due to the influence of respiration.

Description of the Bigeminal Pulse and some of its Modifications.—We have already indicated what is the special feature of the adult type of irregularity. We may now, however, add something more. Traube described this form as the *pulsus bigeminus* or bigeminal pulse (Fig. 71), and believed the second and smaller beat to be due to an exaggerated dicrotic wave. A simultaneous tracing of the left apex-beat entirely disproves this idea (Fig. 72). It is then clearly demonstrated that two ventricular contractions follow each other in rapid succession during the time occupied by the prolonged pulse interval. The little upheaval marked *p* in the tracings is really a second and *early* systolic contraction, and is followed by a long diastolic period. This so-called *premature systole* (*p*) may be represented in the tracing by a very small or by a relatively large wave. In some cases, when the second systole is not sufficiently strong to raise the aortic valves, the elevation is absent altogether, and then the line of descent of the tracing falls steadily to the abscissa. In such a case it may very easily be mistaken

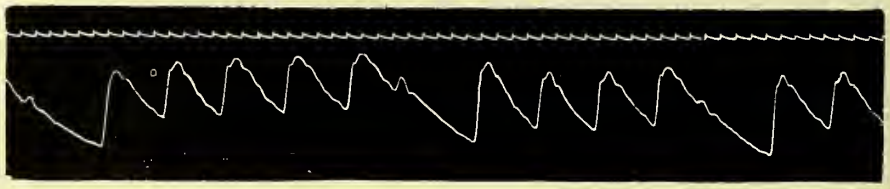


FIG. 71.—The "pulsus bigeminus" of Traube. Male patient æt. 64, suffering from advanced arterio-sclerosis—peripheral resistance enormously increased. ("False ventricular intermission"—Cushny.)



FIG. 70.—Pulse irregularity due to vagus influence. Boy æt. 9 years, under the action of digitalis.

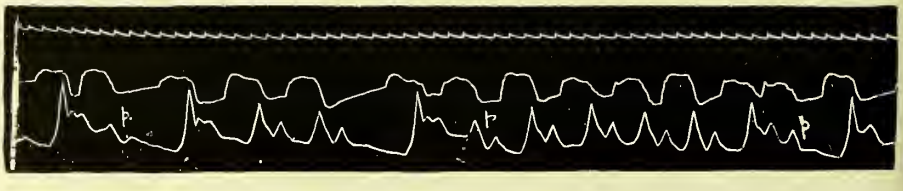


FIG. 72.—The "pulsus bigeminus," with a simultaneous cardiographic record, to show the two ventricular contractions corresponding to the full pulse-beat and the premature beat (*p*). ("False ventricular intermission.")

for the so-called "true ventricular intermission," where the ventricle misses out an entire beat, but the cardiograph or stethoscope will decide the question. The occurrence of this type of irregularity in children is excessively rare, still it is sometimes met with.



FIG. 73.—"Pulsus alternans"—exaggerated development of the premature systole in the bigeminal pulse.

These premature systoles may occur singly at rare intervals, or there may be a series of them following the regular ventricular contraction.

When the systole and the premature systole are of nearly the same size, and there is a long diastolic period between each pair of them, the pulse is sometimes called an alternating one —*pulsus alternans* (Fig. 73); it is simply an exaggerated degree of the bigeminal pulse. When the full systole is followed by two premature systoles we get the so-called *trigeminal pulse* (Fig. 74), and if three abortive contractions follow the full ventricular beat it is a *quadrigeminal pulse* (Fig. 74). After the first full systolic contraction a whole series of premature beats may occur (Fig. 75), and sometimes the entire sphygmogram may be composed of such immature systoles when the pulse is a rapid one.

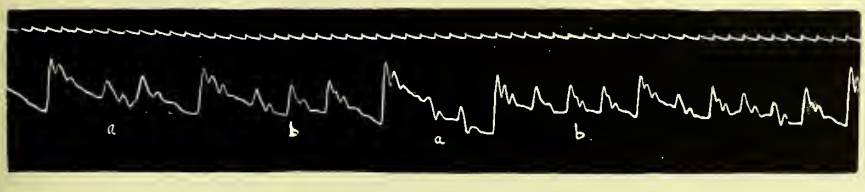


FIG. 74.—The "trigeminal pulse," *a*, *a*; the "quadrigeminal pulse," *b*, *b*.

The bigeminal, trigeminal, and quadrigeminal pulses are usually readily recognised, but sometimes when there is a *regular* irregularity, which occurs after every second, third, or fourth beat, the appearances produced are very similar (*see* Figs. 76, 77, and 78). The long pause, at first sight, seems to be due to a simple prolongation of the diastolic portions of the curve, but a second ventricular beat really occurs during the interval. By referring to the tracings shown in Fig. 79, which were taken from the same patient a few minutes after obtaining Fig. 77, *another* heart-beat is seen to occur during the pause. These sphygmograms tend to support the contention that a *true* ventricular intermission is exceedingly rare, and that in most instances the ventricle really makes a second and premature attempt to contract.

The Strong Pulse-beat after the Irregularity. — After the premature systole there is a long diastolic period, and during this time the arterial pressure falls to a very low point. The left ventricle becomes rather over-filled, and therefore there is almost always a specially strong beat after the irregularity (Fig. 80). In the adult type of pulse irregularity, then, the ventricle — owing, perhaps, to excessive irritability of its muscle—contracts a second time *before* its normal diastolic period is completed. The part of the diastolic phase at which the second contraction occurs is variable, and according to its position so will its character vary.

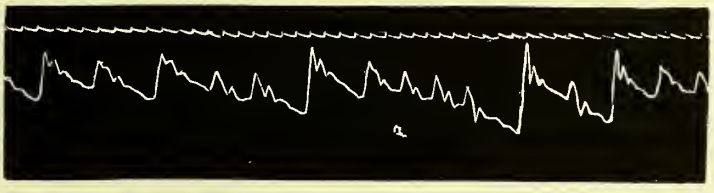


FIG. 75.—Series of premature systoles (*a*).

The Shortened Diastolic Period following the Premature Beat of the Heart. — Sometimes, though very rarely, the premature beat occurs so late that the diastolic period following it is *shorter* than that of the full contraction preceding it (Fig. 81).



FIG. 76.—Regular irregularity after every *second* beat (the second beat may be a very exaggerated premature systole, and then the pulse would be of the ordinary bigeminal type; it is more probable that an early systole occurs during the long pause).

The Cause of the Bigeminal Pulse.—It is very difficult to say why the ventricle in these cases

contracts prematurely. It is not improbable that in some cases a toxic condition of the blood acts as an exciting cause. In elderly patients

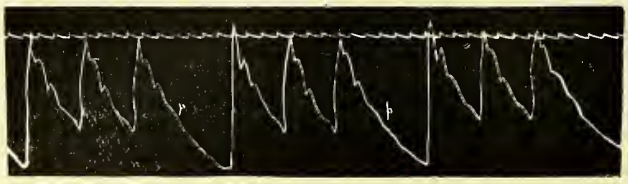


FIG. 77.—Regular irregularity after every *third* pulse-beat; *p* marks the occurrence of a premature systole.

of gouty habit this type of pulse irregularity is very common, and would sometimes seem to be accentuated during the height of an attack. The intrinsic ganglia of the heart may also become unduly excitable, but it is unlikely that the vagus plays any part in the production of this type of irregularity. Stimulation of the vagus slows the heart, whereas the pulse is often rapid when premature systoles occur.

Pulse Irregularities due directly to Respiration.—Before leaving this subject mention may be made of another type of pulse irregularity which is apparently due to respiratory causes. Attention may at present be directed to those irregularities which occur with normal or slightly exaggerated breathing. Those forms which are dependent upon laboured or otherwise abnormal respiratory movements will be discussed in a subsequent section.



FIG. 78.—Regular irregularity after every *fourth* pulse-beat.

The first type of irregularity of this kind is that in which the *inspiratory* movement is specially concerned. When the tracing is examined, it is noticed that some of the pulse-waves in the series are of smaller size than others. The appearance is not quite the same as that of the ordinary form of youthful irregularity already described. I append a tracing from a baby five months old, where this character can be easily recognised (Fig. 82). When a simultaneous respiratory record is made it is found that the shorter waves correspond to the height of the inspiratory phase. Other tracings show the same features, and this irregularity is sometimes present under chloroform anæsthesia, when the respirations are deep.

It is obviously sometimes difficult, if not impossible, to decide in what manner respiration brings about these alterations in pulse form and rhythm. But we may take it for granted that they are not always induced in the same way, and in many cases the action of the vagus in their production is not excited by respiratory influence on the medulla.

More than one pulse-wave in the series may be affected in the manner described by each in-

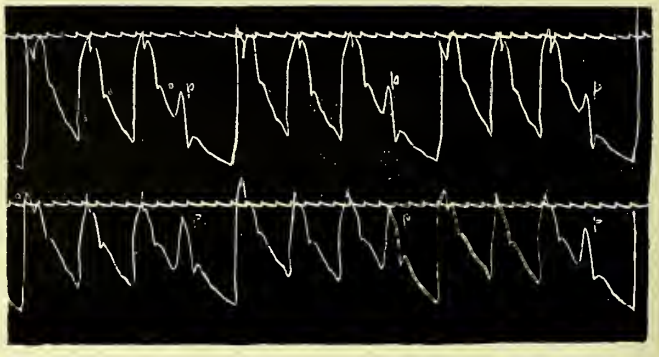


FIG. 79.—Gradual development of the premature systole (*p*) in the case of *regular* irregularity after every third beat. *See* Fig. 77.

spiratory movement, and sometimes the curves occurring during expiration are unusually large. A tracing from a girl, twelve years of age, suffering from chronic rheumatic arthritis, shows these characters, and this form of respiratory modification of the pulse often occurs in children (Fig. 83). The irregularity shown in this tracing evidently bears a close relationship to the respiratory curve; its characters are not quite the same as those of the ordinary type of youthful irregularity.

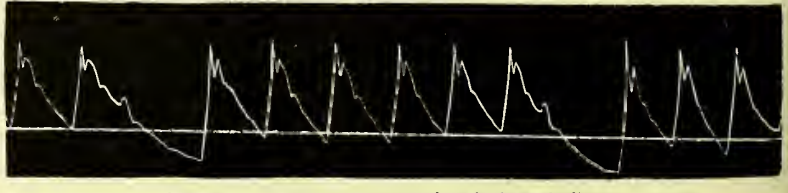


FIG. 80.—The strong pulse-beat after the irregularity.

PULSE AND RESPIRATION

Importance of making Graphic Records of both Events.—Pulse and respiration is a com-

paratively unworked field of research. In the study of this subject under various conditions it is absolutely necessary to make simultaneous records of both events. This can now be accomplished most satisfactorily by means of Mackenzie's "clinical polygraph." This small apparatus is attached to the ordinary Dudgeon's sphygmograph, and it records the respiratory curve on the same strip of smoked paper as that which registers the radial pulse.

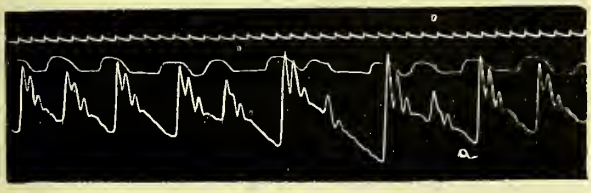


FIG. 81.—The *shortened* diastolic period after the premature beat of the heart (*a*); that shown in the preceding beat is the usual type. The upper tracing is from the apex beat

The Clinical Polygraph.—The following short description of the instrument is taken from Dr. Mackenzie's paper on the "Venous Pulse." It consists of a tambour (A in the figure) supporting the writing lever, the latter being 6 inches in length. From the under-surface of the tambour a pipe protrudes, which is connected by india-rubber tubing with a receiver. The receiver is simply a small shallow vessel, made of lead or some other metal, and is used to receive the impressions from the pulsating artery or vein or from the apex-beat. Screwed tightly to the bottom of the tambour is a stem (B), $6\frac{1}{2}$ inches in length, projecting outward, parallel to the under surface of the tambour. Half an inch of the other extremity of the stem is bent almost at right angles (C), and this portion fits into a slot in the upright stem that supports the movable lever of a Dudgeon's sphygmograph, and this supports the tambour. When the tambour arrangement is adjusted to the sphygmograph the tambour rests with its movable surface vertically, so that the writing lever moves horizontally. The point of the writing lever can be adjusted to any desired place on the recording paper, as there are several movements available at D, E, and F. By this method any pulsatile movement or the respiration can be recorded at the same time, and on the same paper as the radial sphygmogram. Ordinates are made by allowing the levers to write on the paper before starting and after stopping. With a pair of compasses the relative time of the events can be accurately gauged.

Use of the Clinical Polygraph in Children.—The writer has been able to use this little instrument without difficulty, even in newborn infants, either to record the movements of respiration or of the left apex-beat along with the radial pulse. The tracings are admirable, and we now possess a ready and accurate means of studying the influence of the respiratory movements on the circulation.



FIG. 83.—Respiratory irregularity where more than a single pulse-wave is affected (really a pulsus paradoxus). *See* p. 419.

Graphic records of the different types of respiratory movement as found in various states of dyspnœa are of much interest, and these are as yet imperfectly recognised. Clinically, we know that such types exist under conditions of laboured breathing, but the precise relations of the inspiratory to the expiratory phase are not always adequately appreciated. With regard also to the time of occurrence of the *respiratory pause* under various circumstances, much has still to be learnt.

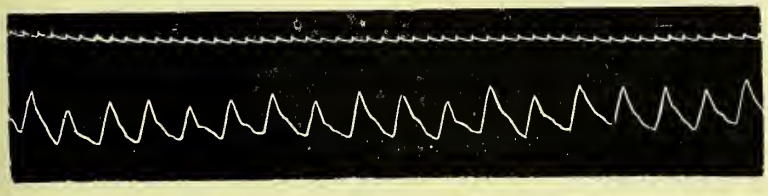


FIG. 82.—A type of irregularity due to respiration in an infant æt. 3 weeks—the smaller pulse-curves correspond to inspiration (normal breathing).

Effects of certain forced Respiratory Movements on the Pulse.—In the dyspnœa of advanced cardiac failure, with the circulation nearly at a standstill, the pulse seldom shows any respiratory variation. Hiccough apparently affects most pulses by causing a temporary arrest of the circulation and a fall of blood pressure, and very deep inspiratory and expiratory efforts with closed mouth and nose—the so-called Muller and Valsalva experiments

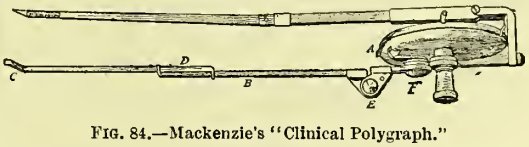


FIG. 84.—Mackenzie's "Clinical Polygraph."

—have also a marked influence. The soft pulse of low pressure is undoubtedly more susceptible to these altered respiratory movements. Pulses of high pressure are not affected to nearly the same degree.

It is certain that the physiological dictum that "during inspiration venous pressure falls and arterial pressure rises, and during expiration venous pressure rises and arterial pressure falls," is absolutely inadequate to explain all the variations of different pulses produced by altered respiratory movements in various conditions. The whole question is one of extreme complexity, and many different influences are associated together to evolve certain results.

Probably the *quantity* and the *quality* of the blood circulating in the vessels of the medulla is the proximate determining cause of the chief variations, through the effects thus produced on the vital centres resident there.

Effects produced by Variations in the Quantity of Blood reaching the Medulla.—During inspiration blood is drawn away from the brain, as is easily demonstrable by watching the sucking in of an infant's fontanelle or the emptying of a full jugular vein. This means a lowering of the blood pressure in the bulbar vessels, and consequently a definite effect is temporarily produced on the control of the circulation exerted by the vagus and vaso-motor nerves.

In the case of the *vagus* centre, diminished blood-supply means *diminished stimulation*, and will lead to an inhibition of its influence in maintaining a normal pulse-rate. Thus in healthy individuals we would expect strong inspiratory movements to cause, in the first instance, a quickening of the pulse. This effect is often produced, but not invariably.

In the case of the *vaso-motor* centre, when its usual blood-supply is diminished, *stimulation occurs*, with the result that its activity soon comes into play in the shape of vaso-constriction and consequent rise of blood pressure.

Effects produced by Variations in the Quality of the Blood reaching the Medulla.—In the case of the *vagus* centre, blood deficient in oxygen or otherwise impure will result in *stimulation*, and the pulse will tend to become slow.

In the case of the *vaso-motor* centre, *stimulation* also occurs, with resulting constriction of the smaller arteries throughout the body and a rise of blood pressure. Thus it will be seen that the combined influence of *diminished quantity* and *impaired quality* of the blood in the medulla will produce, in the case of the *vagus* centre *opposite* effects, and in the case of the *vaso-motor* centre the *same* effects.

An appreciation of these facts may help to assist one in the interpretation of many sphygmograms obtained in cases of spasmodic asthma, and also in other diseases associated with laboured respiration and alterations in the quality of the blood.

The Effects of Afferent Impulses reaching the Vaso-motor Centre—The Depressor Nerve.—Still other influences have to be remembered in dealing with the whole problem of pulse and respiration in pathological conditions. The afferent impulses reaching the vaso-motor centre are of considerable importance, and under certain circumstances give rise to unexpected results. Impulses coming to the centre through most afferent nerves are followed by a reflex rise of blood pressure when the pulse is of low or of medium pressure. But if the vaso-motor centre is actively maintaining a high pressure in the arteries by constriction of their coats, thus inducing abnormally great distension of the heart and embarrassment of its action, then a special afferent impulse to the vaso-motor centre from the heart itself will act in an opposite way, and produce a fall in the blood pressure and relief to the heart.

This is a safety-valve mechanism, and the afferent impulse which brings it into play is conducted through the *depressor* nerve which depresses the centre, thereby producing dilatation of the intestinal vessels by means of the splanchnic nerves.

Action of the Depressor Mechanism in Normal and in Pathological Conditions.—This mechanism for regulating the blood pressure may be frequently called into action in normal individuals, but it is not certain that it always acts so precisely in diseased conditions. During a severe asthmatic seizure, where there may be marked vaso-constriction, and the heart is struggling against undue resistance, relief may be sometimes partially brought about in this manner, and the relaxation of the vessels leads to the lowering of the arterial pressure. When a long inspiration is taken and the breath held, the type of sphygmogram obtained is sometimes suggestive of the same thing—the curves may become larger and more dicrotic during the sustained inspiratory act.

The Connection between the Vagus and Respiratory Irregularities of the Pulse.—A great many of the varieties of respiratory irregularity of the pulse may be explained on the theory already advanced to account for the ordinary type of youthful irregularity. Special emphasis was laid on the fact, that when the pulse temporarily quickened owing to lessened vagus influence, the reaction back to the normal rate was apt to be too extreme. That is to say, the vagus, in regaining its influence, tends to slow down the ventricular beat too much at first, and it does not immediately strike the proper balance with the accelerators. In the ordinary pulse irregularity of the child it is not always the inspiratory act which leads to a temporary diminution of the vagus influence, and therefore a faster pulse. Many other causes operate on the rate of the heart, and some of these may act continuously for a time,

during which the vagus control is overbalanced. This is what happens during *febrile* conditions, and when the temperature falls to normal the vagus reasserts its control, and the characteristic retardation in some of the beats is observed. When a child is kept resting in bed the vagus soon begins to acquire an increased power over the heart, and the irregular rhythm of the pulse is again evident. Ordinary inspiration does not exert a sufficient impression on the vagus centre to affect most pulses. Deep inspiratory movements, however, lead to a certain loss of vagus control and to a quickening of the pulse-rate. This is generally followed by an undue slowing of the next few beats, more or less marked, due to temporary over-action on the part of the vagus when its roots are again stimulated by increased blood-supply (Fig. 85).

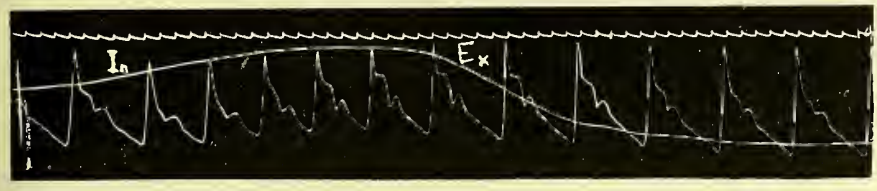


FIG. 85.—Quickening of the pulse with deep inspiration (loss of vagus influence over heart), with slowing at expiration (vagus regaining its influence). The quicker beats are more dicrotic.

Anything, therefore, which leads to a temporary or prolonged overbalancing of the vagus control on the heart will, after its influence is removed, induce a form of pulse irregularity characterised by a lengthening of the diastolic portions of some of the pulse-curves.

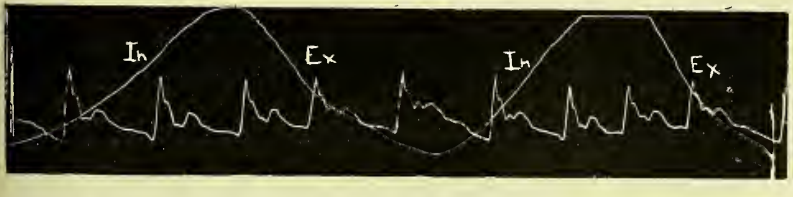


FIG. 86.—Effects of deep respiration in causing pulse irregularity; prolonged beats occur during the expiratory phase after the transient quickening at inspiration. The pulse-curves are more dicrotic during inspiration.

A deep inspiration is only *one* of the many causes which acts in this manner on the vagus centre, and the reaction of this influence is often seen in a prolongation of the diastolic portions of the curves during expiration, for at this period the vagus again regains its control. The tracing, Fig. 86, shows this form of pulse irregularity where respiration is purposely deep, but not abnormally rapid. However, in some cases of laboured and rapid breathing the pulse variations are of a similar character.

The Effects of Deep Inspiration on the Pulse.—It has been already stated that it is the soft, low-pressure pulse which shows the greatest variations with respiration. The effects of forced inspiration on a pulse of this character are to quicken the beats and render the curves more dicrotic. During the time of inspiration, and especially when the breath is held, the pulse-curves often become smaller and are of shorter duration. During expiration the curves become larger, and are generally more prolonged (*see* Fig. 86).

The Effects of Deep Expiration on the Pulse.—Holding the breath at the end of expiration does not seem to produce nearly so marked an effect on the pulse. It generally becomes somewhat slower, and may show a higher pressure. At the end of deep expiration, however, there is often a prolongation of the diastolic periods of the curves.

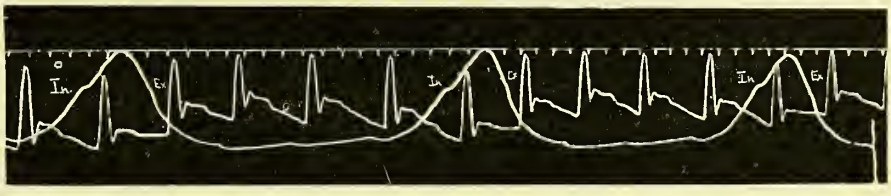


FIG. 87.—The pulse with slow respiration; longer diastolic periods during expiration. At the time of inspiration there is a fall in the tracing. (Large cerebral hæmorrhage, boy æt. 12 years.)

The Pulse with Slow Respiration.—When the breathing is slow and laboured—as in all cases of cerebral compression, tubercular meningitis, tumour, etc.—it is common to have one or more pulse-curves corresponding to the height of inspiration, markedly diminished in size and shortened in duration. The curves occurring at the end of expiration may be somewhat prolonged (Figs. 87 and 88). When the pulse is of high pressure, however, it may show very little respiratory variation (Fig. 89). The *type of respiratory curve* in some forms of cerebral compression is peculiar, and in several cases of simple basal meningitis the writer obtained tracings like that shown in Fig. 90. The respiratory *pause* occurs after inspiration. This occurred also in one case of broncho-pneumonia complicated by pleurisy with effusion, and it

probably does not bear any special significance. In a case of measles ushered in by violent convulsions, with a pulse of great rapidity and a temperature of over 105°, this same peculiar *pulse-wave* during *inspiration* is the *rule* even when respiration is not much exaggerated. Forced inspiration is not always necessary to develop this feature in a child's pulse. With ordinary breathing it can frequently be detected in the sphygmogram.

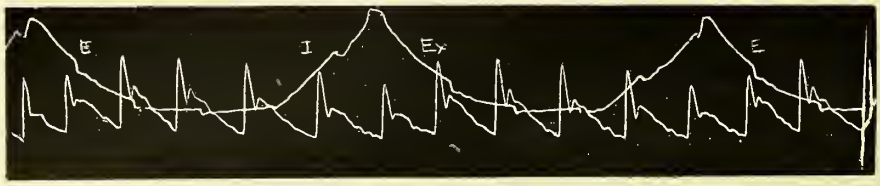


FIG. 88.—Pulse with slow respiration, showing the shorter and smaller curves during inspiration.

One of the best examples of this form of pulse was obtained from a youth who was suffering from a mild asthmatic seizure. There was only slight wheezing present, and the breathing was not really difficult. On feeling the pulse it was found that it periodically varied in volume—so much so, indeed, that by gentle pressure on the artery the impulse seemed at times to be completely absent. Tracings were taken, and the relation of the pulse variation to the respiration was observed. Fig. 92 shows the characters of this pulse, and it is apparently identical in appearance with the so-called "pulsus paradoxus." There was no reason to suspect adhesive mediastinitis here. So this kind of tracing must simply be regarded as an exaggerated degree of the not uncommon diminution of the pulse-wave during inspiration.

type of breathing was found. But this form of respiratory curve does not seem to be present in cases of tubercular meningitis in young children.

The Pulse with Laboured Breathing.—When the respiration is very rapid, as in pneumonia, the pulse-tracing may show a short and a long beat alternately—the former coinciding with inspiration, the latter with expiration (Fig. 91).



FIG. 90.—The type of respiration in simple basal meningitis; respiratory pause occurs after inspiration. (Child æt. 16 months. Pulse 144, resp. 60, temp. 100°.)

Pulsus Paradoxus.—Kussmaul first directed attention to this form of pulse. The most characteristic feature is a distinct depression of the pulse-wave occurring synchronously with inspiration. This peculiar pulse was at one time supposed only to occur in cases where some pleuro-pericardiac adhesion existed. Something of the same kind has, however, been found in other conditions, such as simple pericarditis with effusion, pleurisy with effusion, in mediastinal tumours, and in aneurysm of the thoracic aorta.



FIG. 89.—Very slight respiratory variations in the pulse when the arterial pressure is high. (Tubercular meningitis, child æt. 4 years.)

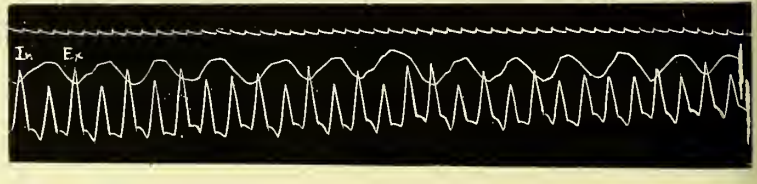


FIG. 91.—Pulse and respiration in pneumonia (fatal type), showing alternate large and small pulse-waves at expiration and inspiration.

Brockbank has called attention to its occurrence in children suffering from dyspnœa due to acute laryngitis.

The writer's own experience is, that in young children this *diminution* of the height of the

Causes of the so-called Pulsus Paradoxus.—In a paper on the "Effects of Forced Inspiration on the Pulse in some Forms of Heart Disease," Irvine discusses this question pretty fully. He

agrees that the occurrence of the pulsus paradoxus may often be associated with pleuro-pericardiac adhesion, but he goes a step farther, and states that a weakening of the heart muscle, due to myocarditis or by effusion into the pericardium, has a share in bringing about this pulse depression.

It is quite certain, however, that the causes already mentioned do not often come into play in the occurrence of this inspiratory variation of the pulse in children.

Brockbank recognises the need of some other explanation to account for the pulsus paradoxus of acute laryngitis in children. In such cases he thinks it is to be found in the great variations of the intrathoracic pressure on the heart and great vessels in the chest cavity. Without going fully into the matter, it may be stated that *difficulty in getting the air into the lungs* is the key-note of his theory—the in-rushing air cannot enter fast enough to fill the expanding lungs. This means that the left ventricle has to pump its contents out of a vacuum into positive pressure.

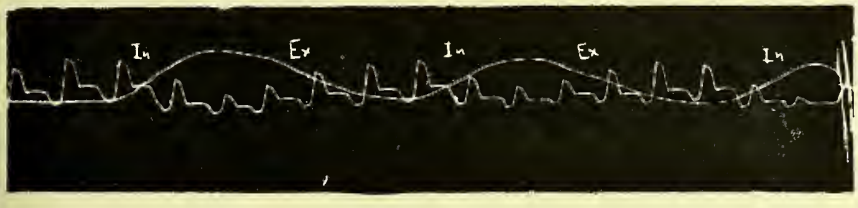


FIG. 92.—Inspiratory diminution of the pulse-curves in a slight case of spasmodic asthma. (Identical in appearance with the true pulsus paradoxus of mediastino-pericarditis.)

Explanation of the common Occurrence of this Pulse Peculiarity in Children. — Brockbank's view may explain why this pulse peculiarity is of such frequent occurrence in young children, even in normal conditions, because the larynx and trachea are of relatively small size. He apparently does not recognise how common this inspiratory pulse diminution is, for he regards its occurrence in acute laryngitis as specially noteworthy.

Extensive employment of the sphygmograph in children leads the writer to the conclusion that this pulse variation is almost invariably met with in all conditions of laboured breathing, whether fast or slow.

Anything which causes obstruction to the entrance of air into the lungs during inspiration accentuates this pulse depression (Fig. 93). Its occurrence has been noted in children who were largely "mouth breathers," owing to enlargement of the tonsils, or in those who were affected with adenoids. Personal experience would therefore lead me to support such a theory as Brockbank advances.

There are, however, certain special features and characteristics of the *pulsus paradoxus*, as seen in cases of mediastino-pericarditis, which distinguish it from the simple diminution of the pulse-wave which occurs so frequently in children with ordinary or slightly exaggerated inspiration under various other conditions.

The writer believes it would be advisable to restrict the use of the term pulsus paradoxus *exclusively* to that type of pulse which occurs in well-marked cases of mediastino-pericarditis; the term should not be applied to any other form of inspiratory diminution of the pulse-curve, however marked it may be. Harris, in his admirable monograph on "Indurative Mediastino-Pericarditis," quotes from Schreiber, who regards the following peculiarities as distinctive of the true pulsus paradoxus:—

(1) Marked diminution in the volume or complete disappearance of the pulse of all arteries, and not only of that of the radial vessels, during inspiration, and especially in the second half or at the end of that act.

(2) The impossibility of replacing the paradoxical pulse by a full pulse by a prolonged and sustained inspiration.

(3) The pulsus paradoxus does not require for its development the inspiration to be deep.

(4) The pulse intermission is accompanied by a regular action of the heart, and the heart shows

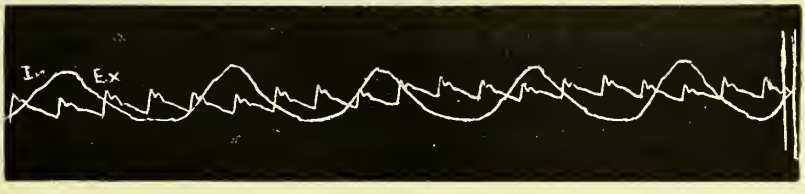


FIG. 93.—Slight degree of inspiratory pulse diminution in a child affected with adenoids.

(5) No weakening of its action during the period whilst an inspiration is being made (four and five might, however, be modified in cases where there is, at the same time, endocarditis or myocarditis).

In addition to the above-mentioned characters Schreiber mentions an associated symptom of great importance—*inspiratory* swelling of the cervical veins.

Diminished Radial Calibre during Inspiration.—In some cases the kind of tracing obtained suggests that the calibre of the radial artery actually becomes smaller during deep inspiration. The pulse-curves during this period seem to indicate that the artery is contracted, and that its

elasticity is impaired. The instrumental apex of the systolic waves is not developed in the smaller curves which occur during inspiration, and this might be taken as evidence that the artery is more fully under vaso-motor influence at this time. The effect of inspiration on the vaso-motor centre in the medulla is to cause stimulation, and it is conceivable that sometimes the arteries are alternately contracted and relaxed as a result of deep inspiratory and expiratory movements.

The Influence of the Blood Pressure in the Venæ Comites Radiales in the Production of Respiratory Variations in the Pulse.—Leonard Hill and others believe that the veins which surround the radial artery bear an important share in the production of many of these respiratory variations of the pulse. There is, these observers state, a mechanical displacement of the button of the sphygmograph above the level of the artery, due to a distension of these veins. In pathological conditions where, by back pressure, the tension in the venous system is greatly raised, this filling of the venæ comites is supposed to account for the various modifications of the sphygmographic tracing which have already been described.



FIG. 94.—The *fall* in the level of the tracing during inspiration is due to mechanical movements of the arm from the participation of the accessory muscles of respiration in the act.

The following passage is quoted from Hill's article on the Circulation in Schäfer's *Text-Book of Physiology*. He says: "The sphygmogram shows variations synchronous with the respiratory movements. Normally these are slight, but they become marked whenever the respiration is either deep or partially obstructed. During deep inspiration the line of the tracing falls, whilst the excursion of the primary wave decreases, and the dicrotic wave becomes more evident. The venæ comites radiales are here emptied, and the expanded lungs swallow up the blood, so that the arterial tension falls slightly. When expiration follows, the line of the tracing rises, and the excursion of each pulse is increased. During a deep inspiration the vessels in the radial sulcus appear to the finger of less breadth and of lower tension. In obstructed breathing the pulse-beats may, during inspiration, almost disappear, for the air cannot then enter freely, and thus the force of the inspiratory movement is thrown largely on to the intrathoracic vessels and the heart."

Results of Clinical Observations on the Pulse during Laboured Breathing.—An examination of the writer's pulse- and respiration-tracings from children and adults shows that the line of the tracing does *not* fall during deep inspiration, except in a very small proportion of cases. In these instances the accessory muscles of respiration were generally called into action, and they probably caused slight movements of the arm which affected the tracings during inspiration (Figs. 94 and 87). By supporting the arm firmly so as to minimise such movements, this kind of so-called respiratory variation disappears.

Thesc observations seem to show that inspiration very often simply leads to a *diminution* of the pulse-curve without lowering the level of the tracing. On the contrary it sometimes happens that there is a *raising* of the level of the pulse-curves during the inspiratory phase (Fig. 95), while the succeeding curves show prolonged diastolic portions coincident with expiration. The diastole of the inspiratory pulse-wave may then be incomplete.

It is very improbable that venous stasis and engorgement really modifies the sphygmogram so generally, or in such a marked manner as Hill asserts. Clinically, in carefully made observations, we need only seriously consider this interference in a very small number of cases.



FIG. 95.—To show the *raising* of the level of the pulse-curves during inspiration.

Hill's Estimate of the Power attained by Venous Pressure.—Hill ascribes enormous power to venous pressure. During the performance of Valsalva's experiment—forced expiration with the mouth and nose shut—he says the pressure in the venæ comites radiales rises to the mean arterial pressure. We do no think this opinion is shared by many physiologists.

Dr. Oliver has failed to detect a direct relation between the evidence of a high radial calibre and that of venous pressure above the average; he has, on the contrary, repeatedly met with low readings of the radial calibre when venous pressure was exceptionally high. An analysis of Hill's tracings (Schäfer's *Text-Book*

of Physiology, vol. ii. p. 105) would seem to indicate that the distended venæ comites are capable of lifting the pad of the sphygmograph through a greater distance, and therefore with a force greater than that exerted by the systole of the left ventricle. It is surely seldom, if ever, that venous pressure can attain this power.



FIG. 96.—Pulse and respiration of a cat under deep ether anæsthesia.

Results of Pulse- and Respiration-Observations on Animals.—The writer has, with the able assistance of Dr. Noël Paton, made some pulse- and respiration-observations on animals, in order to control certain results obtained during laboured breathing in children. The cat was the animal employed. Under ether anæsthesia one of the carotid arteries was fully exposed, and a portion of the artery, separated from the vein, was supported by a piece of wood which was held by a clip attached to a heavily weighted stand. The artery thus lay upon a perfectly rigid bed. A Dudgeon's sphygmograph, with a clinical polygraph attached to record the respiratory curve, was held firmly in a suitable apparatus in such a way that it could be made to rest accurately upon the artery. The trachea was opened and a cannula inserted, through which the animal breathed.



FIG. 97.—Pulse and respiration (cat). Shows the diminution of the pulse-waves at the height of inspiration (fairly deep respiration).

The objcct of these experiments was to ascertain whether the diminution of the pulse-wave, occurring so commonly in children during slightly exaggerated or obstructed breathing, could be obtained when the pad of the sphygmograph rested upon *the artery alone.*

The first tracing, Fig. 96, shows the pulse-curves along with normal respiration under deep anæsthesia. The animal was breathing fairly deeply, and each pulse-wave at the height of inspiration is, even here, slightly diminished in amplitude.

The next tracing, Fig. 97, shows the effects of a series of fairly deep breaths. Now, there is a distinct diminution of each pulse-curve at the height of inspiration. The pen which records the respiratory curve starts a short distance behind that which registers the pulse, so this must be allowed for in the timing of the two events.

Fig. 98 shows the effect of taking three fairly deep respirations. The line of the tracing is slightly *raised* during inspiration.

Fig. 99 shows the effect of taking a long inspiration and then holding the breath. Again a slight *raising of the line of the tracing* occurs, but it is shortly followed by the usual result, namely, the diminished size of the curves. A sufficiently deep breath will practically abolish the pulse-wave, and thus produce an extreme degree of the so-called pulsus paradoxus.



FIG. 98.—Pulse and respiration (cat). The line of the tracing is *raised* during inspiration (respiration is deep).

Another tracing is appended, Fig. 100, which shows the raising of the level of the pulse-tracing produced by inspiration when the animal was dying. The india-rubber bag which transmitted the respiratory movements to the tambour of the clinical polygraph was fixed to the thorax of the animal in such a way that it lay over the cardiac area, and therefore received the pulsations of the ventricle. The animal was at this time only making an occasional sighing respiration. Therefore the record of heart-beats in the tracing is only broken twice by a respiration. Each time, however, when this occurs



FIG. 99.—Pulse and respiration (cat). The effect of a long inspiration with breath held is to raise the level of the tracing and diminish the amplitude of the pulse-curves.

there is a distinct elevation of the line of the tracing.

The Effects of Venous Pressure on the Form of Pulse Tracing are, clinically, unimportant.—It seems clear from these experiments that this common respiratory variation of the pulse—diminution in the size of the beat—is not

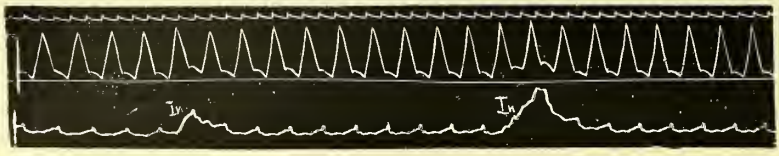


FIG. 100.—Pulse and respiration (cat). The animal is dying, and the lower tracing records the heart-beats, which are twice broken by a sighing respiration (In). The raising of the level of the tracing at inspiration is again observed.

necessarily due to alterations in the pressure of the veins upon which the pad of the sphygmograph may rest at the time the tracing is made.

The writer believes that the force of the inspiratory movement acting on the intrathoracic vessels, and also directly upon the heart itself, plays the chief part in the production of many of these variations of the pulse-wave. In some instances, perhaps, inspiration may induce certain changes in the pulse through effects produced on the vaso-motor centre. Brockbank's explanation of the pulsus paradoxus in acute laryngitis is based upon the effects produced by the expanding lungs on the heart and vessels within

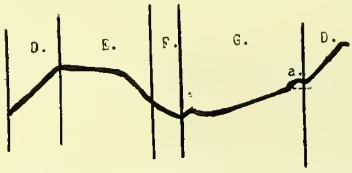


FIG. 101.—Diagrammatic representation of the ordinary cardiogram. Filling of the ventricles from the auricles. *a*, Contraction of auricles.

the chest, and the form of pulse obtained in this condition is simply an exaggeration of the common inspiratory variation of children. Hill recognises this influence of inspiration on the intrathoracic vessels, but asserts that it affects the sphygmogram mainly through alterations thus produced in the pressure of blood in the veins accompanying the radial artery. It is most unlikely, however, when the left ventricular systole raises the pad of the sphygmograph through a certain space, that the venous pressure in the venæ comites is capable of raising the pad through a greater space, and, clinically, the careful observer may practically ignore the participation of the veins in the modification of the sphygmogram.

Heart Movements

The Apex-Beat.—The left apex-beat indicates the chief movement of the heart. It may often be found in the fourth interspace, and outside the nipple line in children, but in adults it is normally well inside this line. When this movement is produced by the left ventricle it is due to a displacement *forwards* of the hardened apex of the heart. During the whole period of systole the apex of the heart is lying against the interspace in the chest wall.

The Component Parts of the Cardiographic Tracing.—The ordinary cardiogram from the left ventricular systole may be interpreted as follows (*see* Figs. 101 and 102):—1. The forward movement of the ventricle as it *begins to contract* (D). 2. Retention of the apex against the interspace, *i.e. full contraction* (E). 3. The backward movement of the ventricle as it *begins to dilate* (F). 4. The gradual swelling of the ventricle up to the *full dilatation* (G). Sometimes a small elevation (*a*) before the period D marks the contraction of the auricles.

The period E may be very transient if the



FIG. 102.—The ordinary cardiogram from the apex-beat of a child æt. 10 years, showing the different phases of the ventricular contraction. The lower tracing was taken simultaneously from the radial pulse. A *rise* in the cardiographic tracing occurs at the time of ventricular systole; E in sphygmogram.

ventricles shrink during their systole away from the interspace (Fig. 103), and it may be represented by a large, well-sustained elevation, the "systolic plateau," when the apical protrusion into the interspace is forcible and lasting (*see* Fig. 104).

The "Shock" due to the Systole of the Left

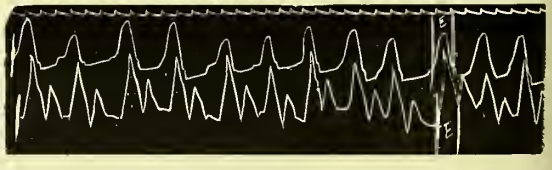


FIG. 103.—Cardiogram where the retention of the ventricular apex against the interspace has been transient—rounded instrumental apex, E, to the curve due to "shock" of systole.

Ventricle.—The *shock* due to the left ventricular systole is important, for it is quite a different thing from the systole itself. The systole lasts during all the time of the apex-beat. The "shock" may often be a transient portion of the cardiogram, but it may jerk up the lever of the instrument and form a large instrumental apex in some tracings, which is sometimes *the only* movement distinguishable.

The "shock" and the apical protrusion may sometimes appear as two distinct elevations, just in the same way as the "systolic wave" in the sphygmogram may appear as a divided summit.

Value of Simultaneous Cardiographic and Pulse Tracings. — The cardiographic tracing when made simultaneously with that of the radial pulse is of great assistance in the interpretation of the various forms of irregularity and intermittency. The impulse from the left ventricle may also often be used to time the occurrence of other movements which are sometimes found over the eardiac area, or such movements may be timed with the radial pulse, which is practically the same thing.

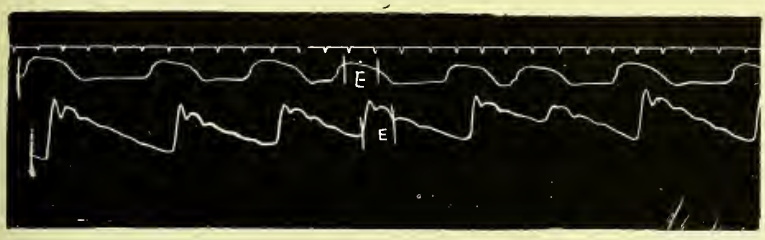


FIG. 104.—Cardiogram to show prolonged retention of heart's apex against the interspace; period E is the "systolic plateau."

Retraction of Tissues near the Heart during the Ventricular Systole.—It is important to remember that when the heart contracts and pushes its apex into the fourth or fifth interspace, it at the same time *draws in* the tissues *round* the interspace. As the heart shrinks during its systole the yielding structures near it *retract*, and thus there is sometimes a *sucking in* of the skin over certain areas at the time of the pushing out of the skin by the left ventricular apex (apex-beat).

We may briefly consider a few of these movements, and their relationship to the cardiogram of the left apex-beat or to the radial pulse.

Epigastric Pulsation. — First with regard to *epigastric pulsation.*

In the acute dilatation of the heart of diphtheria, acute pneumonia, and rheumatism, as well as in advanced valvular disease, one frequently observes very marked pulsation in the epigastric region. This is an important symptom, because it shows that the right side of the heart has enlarged sufficiently to cause a movement of the skin over this area during the systole. Sometimes the skin in this region is *pushed out* during the systole in the same manner as it is in the fifth interspace by the apex-beat, but this is very unusual.

The *usual* movement, when any pulsation is found, is a distinct *retraction* of the skin in this situation at the time of the ventricular systole.

In the dilated heart it is necessary to fully appreciate the fact that the *right* ventricle practically occupies the whole of the eardiac dulness; only a small strip of the left ventricle lies superficially enough to be included in this area. Hence under these circumstances, as Mackenzie has pointed out, what is felt as the apex-beat may not be produced by the protrusion into the interspace of the hardened left ventricular apex. The dilated right ventricle is now occupying this situation, and at the time of its systole, instead of a "pushing out" of the interspace, there is a "sucking in." Thus a *fall* will be produced in the eardiographic tracing at this time instead of a rise when the "receiver" is recording the movement. Should the right ventriele be sufficiently dilated to extend into the epigastrium a similar movement will be obtained in that region at the time of its systole. Here the "sucking in" may possibly be accentuated, because the left ventricle, when it contracts and hardens, tends to pull the right ventricle inwards, that is, away from the epigastrium.

With regard to epigastric pulsation, then, a simultaneous record of the two events, heart and pulse, shows that the systolic portion of the pulse-curve corresponds to a *depression* in the cardiogram, which indicates that the epigastrium has been *drawn in* instead of pushed out at the time of the ventricular contraction (Fig. 105). Mackenzie has called this the "*inverted cardiogram*," and in adults he finds that it is almost invariably due to the systolic contraction of the *right ventricle.* Such a type of cardiogram, if obtained over the usual situation of the apex-beat, shows that the right ventricle is occupying that part of the cardiac dulness, and signifies extreme dilatation of the heart. In children, however, the writer has occasionally found that epigastric pulsation is due to a greatly enlarged left ventricle, and a tracing of a fairly typical cardiogram is obtained (Fig. 106). It is difficult to see how the left ventricle can occupy this situation unless the whole heart is twisted round on its long axis.

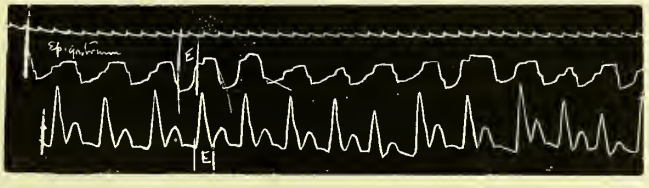


FIG. 105.—The "inverted cardiogram" from the epigastrium. A *depression* in the cardiographic tracing occurs at the time of the systole of the ventricle; E in radial pulse tracing. (Boy æt. 16 with greatly dilated heart.)

Attention has already been drawn to the fact that under treatment, when the dilatation of the ventricle subsides, the epigastric impulse disappears.

Pulsation from the Abdominal Aorta.—The abdominal aorta may sometimes communicate a pulsation to the epigastrium. It is then, of course, a systolic protrusion and not a retraction, and is easily distinguished from the ordinary, or from the inverted cardiogram. Its systolic portion is synchronous with that phase in the radial sphygmogram, and there is no phase corresponding to the period D of the cardiogram —the so-called "pre-sphygmic" period.

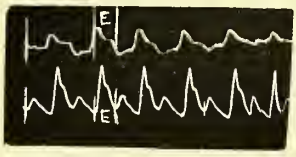


FIG. 106.—Epigastric pulsation from a child aged 9 years, with rheumatic fever and dilated heart; the cardiogram is *not* of the "inverted" type.

Impulse from the Pulmonary Artery.—Among other pulsations seen over the cardiac area mention may be made of that due to the *pulmonary artery*.



FIG. 108.—Simultaneous tracings from the pulmonary artery and radial pulse, showing the correspondence of the systolic portions, E.

Fig. 107 is a tracing taken from a girl ten years old who had a severe attack of rheumatic fever, and the pulsation recorded is evidently directly communicated by the pulmonary artery. There was visible pulsation over the region of the left auricle, and it was doubtful whether it was an auricular movement or one due to the pulmonary artery. Two tracings are given which clearly show that the impulse is not an auricular one, and that the movement is quite synchronous with the systole of the left ventricle and with the radial pulse (*see* Figs. 107 and 108).

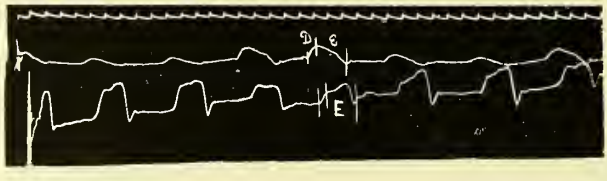


FIG. 107.—Simultaneous tracings from the left apex-beat (upper tracing) and from the pulmonary artery (lower tracing). The movements are synchronous.

Other Movements over the Precordial Region.—Other movements over the precordia are generally due to a retraction of the tissues coincident with the apex-beat. A tracing reveals their true significance when timed with the radial pulse.

Supposed Auricular Pulsation.—A case of special interest may be briefly referred to. A girl aged five years had a congenital cleft in her sternum, just over the site of the left auricle. There was marked pulsation here which was supposed to be auricular. The movement, however, lasted over a longer period than one would expect for an auricular contraction, and it was not altogether suggestive of the pulmonary artery. Tracings of the pulsation were taken simultaneously with the radial pulse. Fig. 109 shows the result. It is a good example of the "inverted cardiogram," and therefore the movement was really due to a sucking in of the tissues above the site of the apical protrusion, or it may possibly have been caused by the systole of the right ventricle.

One sometimes obtains quite a similar tracing, however, off the pulmonary area in children. Fig. 110 is an example; the systolic portion of the radial pulse corresponds to a *fall* in the cardiographic tracing; it is therefore an "inverted cardiogram."

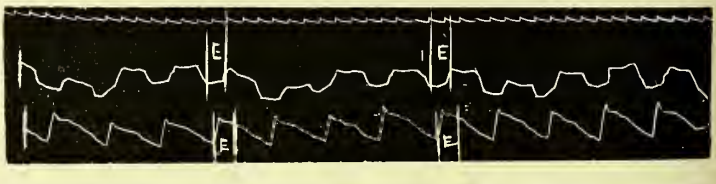


FIG. 109.—Supposed auricular pulsation—really an "inverted cardiogram." Systolic portion of radial sphygmogram E corresponds to a depression in the cardiogram.

Cardiograms obtained directly off the Left Ventricle.—The writer had a unique opportunity, through the kindness of Mr. Dowden, of obtaining cardiograms *directly* off the left ventricle from a child five years old.

A patient under his care suffered from multiple pyæmic abscesses, and one, which formed over the left mammary region, was observed to communicate a cardiac rhythm. It was incised, and pieces of rib were removed. An opening was thus left in the chest wall through which the ventricle, covered by the pericardium and the visceral layer of the pleura, could be easily seen. A small metal "receiver," with rubber tubing attached, was thoroughly sterilised, and was passed through the wound on to the surface of the left ventricle. The other end of the

tubing was connected with the tambour of the clinical polygraph. This was then attached to a Dudgeon's sphygmograph, which was used to make a simultaneous tracing of the radial pulse. As a control observation a tracing was taken by putting the "receiver" on the skin over the site of the apex-beat, and an ordinary cardiogram was obtained. The deep respiratory movements led to some alteration in the characters of the tracing obtained directly off the ventricular surface, but it is quite similar to that made from the apex-beat (Fig. 111). The "receiver" was probably over the apical portion of the left ventricle, and the tracing represents the hardening of the muscle at its systolic contraction—an ordinary left ventricular cardiogram.



FIG. 110.—Tracing taken from the pulmonary area in a child aged 3 years. There is a retraction of the skin over this area (inverted cardiogram) at the time of ventricular systole, E (radial pulse).

Direct Mechanical Stimulation of the Heart under Chloroform.—While the child was under chloroform the heart was touched with the point of the finger, and also with dressing forceps. Direct stimulation applied in this way had no effect whatever on the ventricular beat. The slightly smaller pulse-waves seen in the tracing correspond to inspiration.

The Pulse in Tubercular Meningitis

There are certain diseases in which the characters of the pulse, when carefully studied by means of the sphygmograph, bear some diagnostic or prognostic significance. In the present article the special types associated with tubercular meningitis and pneumonia will be shortly considered.

The Pulse of Tubercular Meningitis in Young Children.—At a certain stage in the course of tubercular meningitis a slow, irregular pulse is often found, and it is regarded by some as a symptom of considerable value in diagnosis. In children under two years of age, however, a markedly slow irregular pulse is not often observed. The writer has gladly availed himself of Dr. John Thomson's experience in this disease. In a series of 18 cases examined by Dr. Thomson, occurring in children whose ages ranged from a half to two years, the pulse was, in 3 of these only, under 90 per minute, and irregular in rhythm. But irregularity was sometimes noted when the pulse was much more rapid.

Irregularity of the Respiration in Tubercular Meningitis.—During the early period of tubercular meningitis in children under two years of age very irregular breathing often coexists with a regular rapid pulse. As an early symptom in these cases, *marked irregularity of the respiration* may be a suggestive and important sign—it is more significant, and more to be relied on in diagnosis, than mere irregularity of the pulse.

In children over two years of age, however, one undoubtedly often meets with a rather characteristic pulse in the early stages of the disease. We say *rather* characteristic, because as a single symptom we do not think it can often be absolutely relied upon for purposes of diagnosis.

Irregularity of the Pulse in Tubercular Meningitis.—Most authorities speak of this pulse as an *intermittent one*; and if one could admit the correctness of some of the text-book descriptions, then the features possessed by it would indeed be sufficiently characteristic to distinguish it from almost any other. So far as the writer's observations go, he does not think the mere *irregularity* means much, because to the finger it is precisely similar to that of the usual type of youthful irregularity already described.

The *slowness* of the pulse, occurring at a time when the temperature is elevated, often to a considerable degree, is significant, but of still greater suggestiveness is the *contracted* state of the radial artery. This, in the writer's opinion, is a very striking condition, and it is an unusual one in young children. There is sometimes a very marked degree of contraction present, and the artery can be rolled like a piece of whip-cord under the finger. The pulse is always slow and hesitating when the vaso-constriction is very pronounced, for the ventricle is then acting against greatly increased peripheral resistance. In this disease slight elevation of the temperature does not always lead to relaxation of the arteries, so the pulse-rate does not quicken. Hence the suggestive triad of symptoms—pyrexia, slow irregular pulse, and contracted vessels.



FIG. 111.—Simultaneous tracings *directly* off the left ventricle and from the radial pulse; the "systolic plateau" E corresponds in time to the systolic portion of the sphygmogram. The respiratory movements have modified the cardiographic tracing.

General Use of the Term "Intermission" in describing this Form of Pulse.—In the description of this pulse irregularity hitherto, the term "intermission" has generally been used. It has already been pointed out, in the classification of pulse irregularities, that the term intermission should only be employed when it can be definitely proved that a whole heart-beat is entirely missed out. There is no absolute certainty that this *true* ventricular intermission occurs in man, except on very rare occasions; and the writer has never met with it in children, nor, so far as he knows, has any one else. By trusting to information derived from the use of the finger alone, the pulse in tubercular meningitis has been called "intermittent" in the sense that an entire heart-beat is, from time to time, omitted altogether. Thus Eustace Smith says, "a slow, irregular pulse, *especially if the intermission be complete*, very greatly increases the value of the symptoms with which it may be associated. It must, however, be remembered that during convalescence from acute disease the pulse in a child often becomes slower and irregular, and may even *completely intermit*, especially during sleep." In another place he states that "the irregularity in rhythm is particularly well marked, because, as a rule, *the intermission is complete*. This is the more evident on account of the slowness of the pulse. Several beats strike the finger in regular succession, and then suddenly *one beat is entirely omitted*, and there is a pause until the rhythm is again taken up by the succeeding beat."

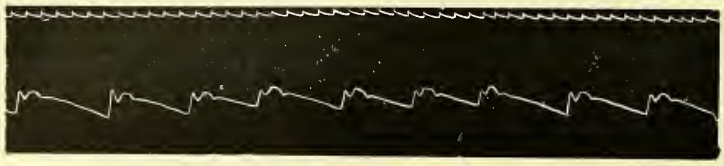


FIG. 112.—Pulse in tubercular meningitis (child æt. 3¼ years). The radial artery is small and tightly contracted. Shows type of irregularity and occasional appearance of anacrotic curves.

The italics are mine, and I would emphasise the fact that this authority assumes the omission of a whole cardiac cycle during the long pulse-pause. This suppositson has been accepted as being the correct definition of what actually occurs.

The General Type of Pulse Irregularity in Tubercular Meningitis.—Sphygmographic examination of the pulse, however, lends no support to such a view, for the so-called "intermissions" are found to be due to a prolongation of the diastolic portions of some of the curves, of the same nature as characterise the ordinary type of youthful irregularity.

Frequent instrumental examination of the pulse in tubercular meningitis has convinced the writer that this is an accurate statement. There is no essential difference, appreciable to the finger, between the type of irregularity found in the early stages of this disease and that which may be found in any healthy child whose pulse is slowing down from any cause.

Other cerebral lesions—simple meningitis, cerebral tumour, and abscess—may sometimes very closely resemble tubercular meningitis, and in order to make a distinction it is often necessary to examine each symptom in detail.

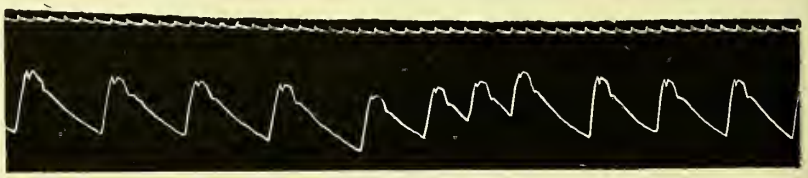


FIG. 114.—Pulse in tubercular meningitis (child æt. 2½ years). Radial artery cord-like. Shows the rather characteristic type of irregularity in this disease. (Anacrotic curves.)

Under such circumstances the sphygmograph may reveal certain pulse features which could not be ascertained without its aid, and which may be of considerable value in differential diagnosis.

A Special Feature in the Pulse Irregularity of Tubercular Meningitis.—In a typical case of tubercular meningitis, if the sphygmograph be used every twelve or twenty-four hours throughout the early stages of the disease, one may sometimes secure a tracing where the pulse-waves exhibit one particular feature which is not to be observed in sphygmograms obtained from most other cerebral affections (intracranial tumour excepted) for which it may be mistaken. The feature referred to is somewhat difficult to describe, but an examination of the tracings (Figs. 112-115) will clearly show the peculiarity to which it is desired to draw attention. In some of the curves the length of the systolic wave is shortened, and the second summit of this wave then rises to a higher level than usual

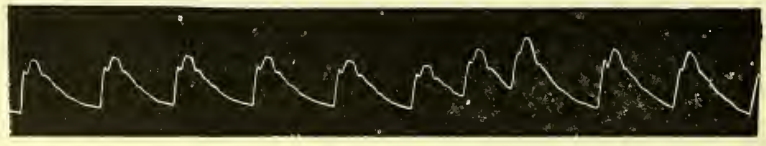


FIG. 113.—Pulse in tubercular meningitis (child æt. 2½ years). Radial artery contracted. (Anacrotic pulse.)

above the first summit. This is equivalent to saying that occasionally the curves in the tracing become of the anacrotic or bisferiens type. The radial artery is always tightly contracted when such tracings are obtained, and the appearance of the sphygmogram suggests that the calibre of the vessel is undergoing variations, from vaso-motor influence, even while an observation is being made (*see also* Figs. 16 and 28). It cannot be certainly affirmed that this is a feature *peculiar* to the pulse of tubercular meningitis, or that it can be demonstrated in every case of that disease. When such a tracing is obtained, however, it is noted that it differs in this respect from the usual type of youthful irregularity. Otherwise the two forms of irregularity would be precisely similar.

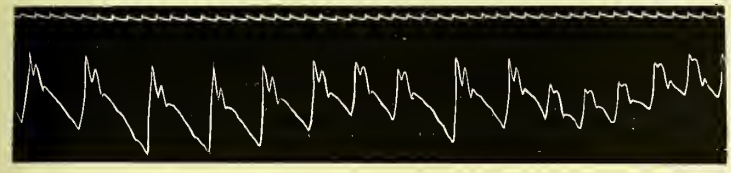


FIG. 115.—Pulse in tubercular meningitis (child æt. 2½ years). The radial artery is not so tightly contracted—temp. 100·2°. The pulse-curves are now of the bisferiens type, but the same type of irregularity is shown.

Changes in the Character of the Pulse during the Course of Tubercular Meningitis.—In some cases of tubercular meningitis, perhaps the remarkable variation in the character of the pulse-curve from day to day, or even from hour to hour, is the most striking thing noted. This is another suggestive point in the differential diagnosis of the disease. The pulse may be fairly regular, and may alter while it is being felt into a halting, irregular rhythm. Sphygmograms of the type shown in Figs. 116 and 117, where a series of large and small beats occur alternately throughout the tracing, are sufficiently remarkable to attract attention. One is tempted to believe that such appearances are of instrumental production, but this is not the case. When the slow, irregular pulse of tubercular meningitis is quickened from any cause, it at the same time becomes regular, and in the final stage of the disease becomes enormously rapid. During this period the vagus never, even temporarily, regains its control over the heart, and the pulse is *constantly* fast (Fig. 118).

During the early stages of the disease, when the pulse is slow, the sphygmogram is invariably one of the high-pressure type, even though the wave is excessively small and feeble. In the later stages of the disease, however, when the pulse becomes rapid, the pressure may fall, and the tracing is sometimes dicrotic.

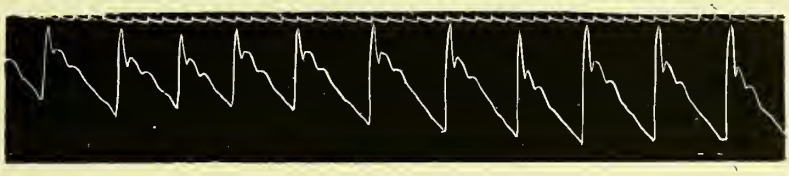


FIG. 117.—Pulse in tubercular meningitis (child æt. 3¼ years). To show the striking variations in the amplitude of the pulse-curves.

With regard to the pulse in simple basic meningitis there appears to be nothing specially characteristic. It is commonly fast from the onset of the disease, which is generally sudden. Many of the children attacked are under two years of age, but even in older children the writer has never obtained tracings which presented any similarity to those of well-marked tubercular meningitis. It is often of fairly high pressure, and the form of the curve may not always alter under slight pyrexia (Fig. 120); but there is never the same remarkable contraction of the artery, and the pulse is at no time either so slow or so markedly irregular as in the tubercular variety. The form of respiratory curve obtained in some cases of simple basic meningitis has already been referred to as a point to be considered in differential diagnosis.

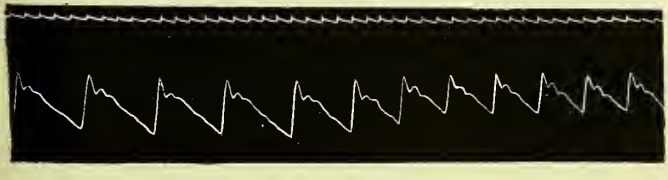


FIG. 116.—Pulse in tubercular meningitis (child æt. 2¾ years). To show the changes in pulse form in the course of a single tracing.



FIG. 118.—Very rapid pulse at the final stage of tubercular meningitis (child æt. 2$\frac{9}{12}$ years).

The Pulse in Pneumonia

I. The Pulse in Croupous Pneumonia.—The pulse in croupous pneumonia in early life

shows some characters which are not usually observed in children. Attention has already been drawn to the interesting fact that high temperatures in infants under one year of age do not render the pulse dicrotic. Therefore sphygmograms which are taken from babies suffering from acute croupous pneumonia indicate relatively high pressure, and one must not infer from this that the general state of the heart is satisfactory, and that the child is doing well (Fig. 121).



FIG. 119.—Very rapid pulse (160 per minute) showing respiratory variations from a child aged 2½ years with tubercular meningitis. Temp. 100°, resp. 48. (Upper tracing is of respiration.)

In children over one year of age, and under three years, tracings are sometimes obtained which indicate high pressure, and sometimes the pulse-curves are of the anacrotic type. But as a rule, in young children, sphygmograms taken at the onset of croupous pneumonia only show high-pressure characters over a comparatively short period. In a day or two, or sooner, the systolic wave appears as a single summit, and the dicrotic notch is well marked.



FIG. 120.—Pulse in simple basic meningitis (child æt. 3½ years). Shows fairly high pressure curves under slight pyrexia (temp. 100°), but is rapid and fairly regular. No contraction of radial artery.

In moderately severe cases of croupous pneumonia in young children the type of pulse-curve at the height of the disease is of the character shown in Fig. 122.

The State of the Arterial Blood-Pressure.—The onset of acute croupous pneumonia in children does not seem to be characterised by a full, bounding, high-pressure pulse, as is often the case in young adults. The writer has made numerous observations on the state of the arterial blood-pressure in the pneumonias of children by means of the large Hill-Barnard sphygmometer; sometimes it is fairly high at the very commencement of the disease in children about five years of age and over, but only for a comparatively short period (Fig. 123); it soon falls, and tends to remain low.



FIG. 122.—Typical pulse of croupous pneumonia at the height of the disease (child æt. 3½ years).

The Relation of the Blood-Pressure to the Post-mortem Appearances of the Lungs.—In croupous pneumonia of the so-called sthenic type, in healthy young adults, it is not unlikely that the greatly increased arterial blood pressure at the invasion of the disease leads to an exudation of fibrin and corpuscles into the air vesicles of the lung. Professor Hamilton, from his large experience of post-mortem examinations, states that the lungs of young children who die from so-called acute croupous pneumonia do not present the same features in this respect as the lungs of adults who die of the same disease. Ten years ago he remarked to the writer that the relatively low blood-pressure in children during pneumonic attacks might partly explain why there was no throwing out of fibrin into the vesicles of the lungs. Observations on the pulse- and blood-pressure in acute croupous pneumonia in children tend to confirm this opinion.

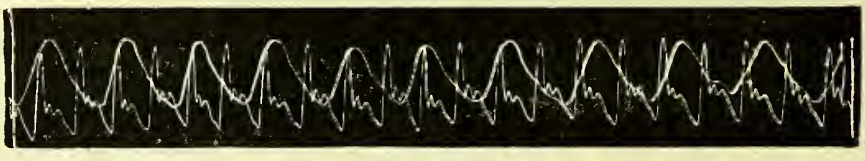


FIG. 123.—Simultaneous tracings of pulse and respiration in a typical case of acute croupous pneumonia at the commencement. Shows good arterial pressure. (Child æt. 8 years. Temp. 102°, pulse 120, resp. 60.)



FIG. 121.—Pulse in croupous pneumonia; child 11 months old (relatively high pressure).

The Special Tendency to a Fall of Blood-Pressure in the Pneumonias of Children.—One of the most striking points about pneumonia of all kinds in children is the remarkable tendency for the blood pressure to fall, and in *broncho-pneumonia* this is the main thing to guard against. To anticipate the, sometimes fatal, fall of blood pressure is the chief object of our treatment.

Prognosis in Croupous Pneumonia.—In croupous pneumonia the main thing to note with regard to prognosis is that in favourable cases the pulse pressure increases very shortly after the crisis occurs. The pulse then slows, and the youngest patient who showed such a pulse was a child two years old, and it was at the onset of an attack of apical lobar pneumonia (Fig. 127). The temperature was 102°, and the pulse 130 per minute, but the child was not much distressed. In a few days, after a good crisis, convalescence set in, and the pulse became slow and regular. The cause of the peculiar heart-rhythm was inexplicable, but it did not bear the same evil significance as in Dr. Mackenzie's cases.



FIG. 124.—Pulse in croupous pneumonia shortly after the crisis in a favourable case. (From the same patient whose pulse is shown in Fig. 122.)

the youthful type of irregularity appears (Fig. 124). This slow, irregular pulse may continue for many weeks during convalescence.

In unfavourable cases the pulse-tracing indicates a falling pressure and becomes very rapid. The pulse-waves are of unequal size, and the dicrotic wave may disappear before death (Fig. 125).

The significance of pulse irregularity in relation to the prognosis of croupous pneumonia is an interesting matter. An elderly patient may habitually exhibit an irregular pulse; he takes pneumonia and becomes feverish. Under such conditions, according to Mackenzie, his pulse should become more regular; if it does not tend to do so the prognosis is bad. Another of Mackenzie's observations is of great practical importance. He states that in young adults suffering from lobar pneumonia with a temperature of 103° or over, the occurrence of pulse irregularity before the crisis is an invariably fatal sign. In these cases the pulse is a very rapid one, and the irregularity is either of the so-called "false ventricular" or "false auricular" type (Fig. 126). The "premature systole" may only occur at long intervals. In adults Mackenzie has also noted the fatal significance of a pulse which, shortly after the initial rigor, exhibits an absence of the dicrotic wave.



FIG. 126.—Pulse *of bad prognosis in* croupous pneumonia in the adult, before the crisis. This type of pulse, the occurrence of irregularity in the early stage of the disease, is generally of fatal significance.

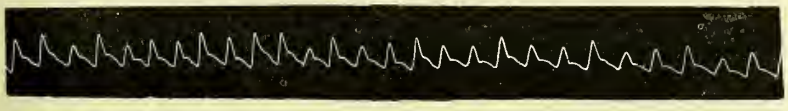


FIG. 125.—Pulse in croupous pneumonia in an unfavourable case. (Child æt. 2¼ years. With temp. , pulse 160, resp. 84.)

A pulse which shows the "premature systole" type of irregularity is, as already stated, excessively rare in children, yet, curiously enough, the febrile pulse of young babies is most unusual, and the fact that it is found in broncho-pneumonia supports the belief that the blood pressure tends to fall unusually low in this disease. Observations with the sphygmometer seem to show that the temporary initial rise of pressure sometimes noticed in cases of croupous pneumonia does not occur in broncho-pneumonia. The pulse-curve in this disease—except in infants under a year of age, for the reasons already mentioned (*see* p. 405)—invariably indicates low pressure.

The Type of Pulse-curve in the Early Stages of Broncho-pneumonia.—The kind of sphygmogram obtained in the early stages is more markedly dicrotic than in the croupous variety (Fig. 128). In cases which are going on to a favourable termination the pulse pressure shows indications of increasing, the pulse slows, and a prolongation of the diastolic periods of some of the curves is noted.

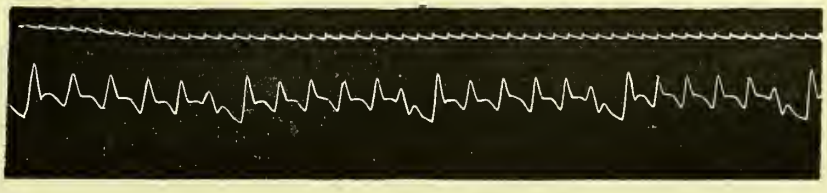


FIG. 127.—The same type of pulse as shown in Fig. 126, but occurring in a child æt. 2 years with a mild apical pneumonia; had no evil significance.

II. THE PULSE IN BRONCHO-PNEUMONIA.—Sometimes in infants under one year of age, suffering from this disease, a dicrotic pulse may be found when the temperature is high. Dicrotism in

But in those cases which progress steadily to a fatal issue there is a type of pulse-tracing to be obtained which is highly characteristic. The pulse is exceedingly rapid, from 140 to 160, and the respirations are about 70 or 80 per minute. The pulse-waves are of various shapes, and show varying degrees of pressure, but the most important feature is the more or less complete disappearance of the dicrotic wave (Fig. 129). This is a sure indication that the blood pressure has fallen to a dangerously low point.

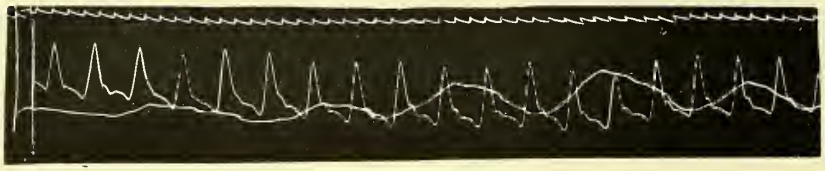


FIG. 128.—Typical pulse of broncho-pneumonia in the early stages of the disease (child æt. 7 years). Indicates lower blood-pressure than that of the croupous variety.

Fatal indications are afforded by this type of sphygmogram, especially if it be obtained in the early stages of the disease, and if it persists for several days. On several occasions this pulse feature has been observed in the broncho-pneumonic attacks of children at the height of the disease, but it was then quite a transient condition. Sometimes, however, this kind of pulse is found at a time when the child is apparently not very ill, and it persists day after day. The writer has never known recovery to take place under such circumstances. On one occasion the fatal event was predicted a fortnight before its occurrence, at a time when the child seemed to be in a fairly satisfactory condition, to judge from other symptoms. Therefore these sphygmographic appearances of the pulse in broncho-pneumonia are of great significance in prognosis, and this particular form of pulse-tracing may fairly be regarded as a *type* which experience has proved to be of decidedly bad omen.

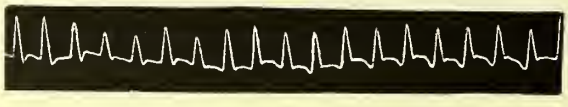


FIG. 129.—Pulse in broncho-pneumonia of fatal significance if it persists for several days (child 1¾ years old).

One other point about this pulse is that the systolic wave may be either *pointed* or *rounded*, and the *sharper* the apex of the tracing the *better* the prognosis. The worst possible type of curve is that in which there is a blunt-pointed apex and a wave of very small amplitude. The pulse-curves are also of unequal size (Fig. 130). These are indications that the heart-muscle is failing, and probably that dilatation of the organ is commencing. Shortly before death in broncho-pneumonia a pulse-tracing showing these characters is often seen. Very similar tracings are obtained in cases of late mitral stenosis (Fig. 131), and also in children dying from auto-intoxication from the intestinal canal.

There is a persistent tendency to failure of the blood-pressure in severe attacks of pneumonia in children. In the catarrhal variety this is especially the case, and if the fall is not arrested very grave symptoms result. In these cases the great lowering of the blood pressure seems to be primarily due to an intense relaxation of the blood-vessels all over the body. The general vaso-motor tone seems to be entirely lost. This may possibly be due to a special toxin. The condition of the circulation seems to be somewhat similar to that found in a well-marked case of exophthalmic goitre, or the same state might be produced by great overdosing with thyroidin. In cases of severe broncho-pneumonia in children the myocardium may suffer, partly from insufficient nutrition, owing to inadequate blood-supply through the coronary arteries, consequent upon the lowered blood pressure; and if we could counteract this extreme relaxation of the blood-vessels we should be fulfilling one of the first indications of rational treatment.



FIG. 130.—The worst type of pulse in pneumonia; pulse-curves of unequal size with *blunt* apices (child æt. 9 years).

In this connection suprarenal extract may possibly be worthy of a trial, and a sterilised decoction of the medulla of the gland might be injected into a superficial vein by means of a hypodermic syringe. Or adrenalin might be used. These cases are so fatal in young children, and chiefly so from cardiac failure, that one would gladly welcome any new measure which could be adopted with a fair prospect of success.



FIG. 131.—Same type of pulse as that of fatal pneumonia, from a case of late mitral stenosis.

VENOUS AND LIVER PULSATIONS

Until within recent years our knowledge of these forms of pulsation has been of the most

meagre and unsatisfactory description; even the main features of the ordinary jugular pulsation had been misinterpreted both by clinicians and experimental workers. In 1896 Mackenzie published his luminous papers on "The Venous and Liver Pulses," wherein is to be found a full and lucid description of the whole subject. His researches stand alone, not only in the numerous new facts recorded, but also in the interpretation of them. In this article the writer is indebted to Dr. Mackenzie for permission to give a short summary of some of the main facts of interest in the study of the venous and liver pulses. For fuller information Dr. Mackenzie's recently published work must be consulted.

The right heart frequently dilates, and thus permits blood to regurgitate back into the auricle during the systole of the ventricle. The tricuspid valves are readily rendered incompetent, and the older physicians used to speak of them as "safety valves," because over-distension of the right ventricle was in this way naturally relieved. But incompetence of the tricuspid valves is not always associated with a pulse in the jugular veins, and the explanation of this fact is important, because herein is found the clue to the true interpretation of the various forms of venous pulsation. The explanation is that *the dilating auricle is interposed between the ventricle and the veins.*

From a study of the venous pulse we obtain information regarding the condition of two chambers of the heart, the right auricle and ventricle; the arterial pulse only indicates what the heart is doing during the time of the ventricular contraction. In cases of pulse irregularity the venous pulse reveals what is occurring in the various chambers of the heart during its irregular action, and forms the only accurate means of diagnosing the kind of arrhythmia present. Pulsation in the jugular vein is found in various forms of functional heart failure, in chlorosis, pregnancy, pernicious anæmia, etc.; but in such cases, however great the heart failure may be, there is never pulsation of the liver. The liver pulse occurs only when there is dilatation of the right heart secondary to mitral disease, and then the disease is always far advanced.

In the jugular vein near its junction with the subclavian there are two valves which in normal conditions prevent regurgitation of blood into the neck. During auricular systole the circular muscular fibres of the venæ cavæ, where they join the auricle, contract with sufficient power to act in the place of valves, and thus prevent regurgitation of blood into the veins. Distension of the right heart impairs the functions of these circular fibres, and a wave of blood is thus sent backwards with each auricular systole. The jugular valves are still between the contracting auricle and the vein in the neck, but they are readily rendered incompetent in some patients. When this happens a wave of blood is sent up the vein into the neck, due to and synchronous with the auricular systole.

The vein, after being filled, collapses as the auricle passes into diastole, and this collapse is the movement usually observed when the neck is inspected; it is found to correspond to the systolic portion of the arterial pulse.

If a graphic record be taken from the vein by means of the clinical polygraph, the movements can be accurately timed with the apex-beat, carotid, or radial pulse, and the causes of the various events can usually be easily recognised. Thus a wave preceding the carotid pulse can only be produced by the systole of the auricle. In like manner, when a wave occurs in the veins synchronous with the carotid pulse, it can be referred with certainty to the systole of the right ventricle. When the tracing shows a wave due to the auricular systole it frequently happens that there is also a wave due to the systole of the ventricle. The time of occurrence of the ventricular wave is delayed for a longer or shorter interval after the carotid pulse.

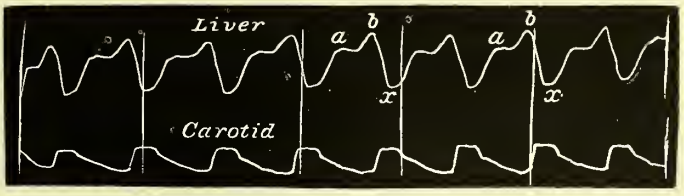


FIG. 132.—Tracing of an auricle liver pulse taken at the same time as the carotid pulse to show the large wave (*b*), which is due to the auricular systole. (Mackenzie.)

The venous pulse may thus be divided into two forms, viz. the auricular venous pulse and the ventricular venous pulse. The first is that form which presents distinct evidence of the functional activity of the right auricle. In this form there is also a wave due to the ventricle, and as it increases the auricular wave decreases and finally disappears, and thus the form called the ventricular venous pulse is developed. The ventricular venous pulse is but a more advanced stage than the auricular; and as during its development there is a gradual fading of the auricular wave, there is a period when such terms do not sufficiently denote the character of the pulse. But the terms are convenient for descriptive purposes. Similar types of pulses may be recognised in the liver.

In Fig. 132 simultaneous tracings are given of the liver pulse and the carotid. The liver pulse is given in place of the jugular, as there are other movements communicated by the carotid, owing to its proximity to the vein, when taking a tracing of the jugular pulse.

When there is a liver pulse it is always identical with the jugular pulse, the blood being sent from the auricle through the inferior vena cava. In this tracing it will be seen that there is a large wave (*b*) which immediately precedes the carotid pulse. At the same time that the carotid pulse appears, the wave (*b*) is falling, and the carotid pulse therefore corresponds with the collapse of the vein or liver. The wave (*b*) can only be due to the auricular systole.

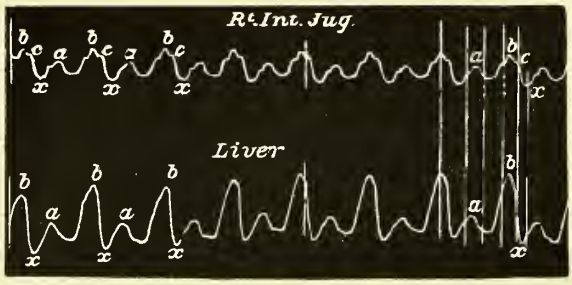


FIG. 133.—Simultaneous tracings of the venous and liver pulses, showing the wave (*c*) in the jugular pulse which is really due to the carotid impulse. (Mackenzie.)

In Fig. 133 there is a simultaneous tracing of the liver and jugular pulses. In the jugular pulse there is a small wave (*c*), which interrupts the fall of the auricular wave (*b*). This small wave (*c*) is really due to the carotid. When the carotid is very small and the jugular pulse very large, the tracing may not show this carotid wave. In Fig. 134 the carotid pulse is taken at the same time as the jugular, and the wave (*c*) is found to correspond exactly with the time of the carotid pulse. The wave (*c*) has, of course, nothing to do with the venous pulsation; it is simply due to the movement of the carotid artery in the neck (*see* p. 398).

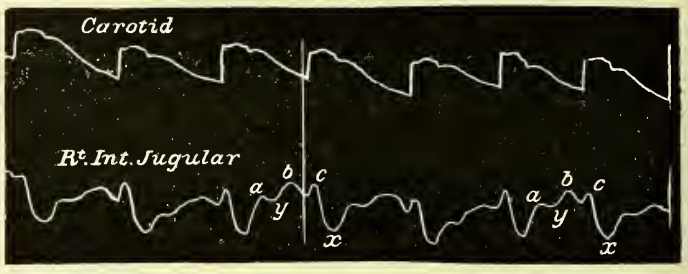


FIG. 134.—Simultaneous tracings of the carotid and jugular pulses, showing that the wave (*c*) corresponds exactly with the time of the carotid pulse. (Mackenzie.)

Immediately following the auricular wave (*b*) there is a great collapse of the vein—(*x*) in the tracings. This is due to the sudden emptying of the vein into the auricle during the auricular diastole, and it is synchronous with the arterial pulse. This fall is interrupted by a second wave (*a*), which is called the ventricular wave. Mackenzie has clearly demonstrated the true cause of this wave, and it is due to the ventricular systole. It appears a little later than the carotid pulse, because the first part of the wave is absorbed by the dilating auricle. This is the important factor in the interpretation of the venous pulse; it is not till the auricle has filled that this wave appears.

When this explanation is fully appreciated it is easy to understand how a venous pulse of the auricular type becomes converted into the ventricular form—how the ventricular wave enlarges when the auricular diminishes. The powerful right ventricle pumps blood into the thin, weak-walled auricle until the latter becomes enormously distended. Matters proceed until complete paralysis of the auricle occurs, and, according to Mackenzie, this is by no means a rare event, though it is not properly realised by observers. Before this stage is reached the auricle is still capable of contracting, and Fig. 135 represents a tracing where the liver pulse is taken at the same time as the radial pulse. The auricular waves (*b*) and the ventricular waves (*a*) are now about equal. A subsequent tracing from the same patient (Fig. 136) shows only one wave, and that wave is entirely ventricular systolic in time. There is absolutely no sign of auricular contraction. Dr. Mackenzie made a post-mortem examination on this case a month later, and found that the auricle resembled a thin-walled sac with a few strands of muscular fibres scattered over it. Patients who exhibit a venous pulse of this kind, a pulse demonstrating that the auricle has ceased to contract, may apparently live for many years. The auricle is then merely a reservoir for the blood during the ventricular systole.

A venous pulse of the auricular type may not signify much cardiac failure; but if the liver pulse is also present, then it is a sure indication of advanced heart mischief. The auricular venous pulse may be temporarily present when there is little deviation from the normal standard of health.

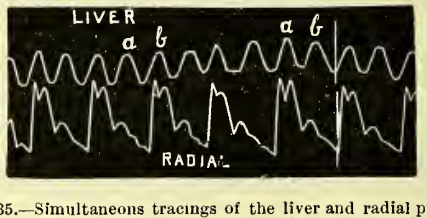


FIG. 135.—Simultaneous tracings of the liver and radial pulses. The liver pulse shows auricular (*b*) and ventricular (*a*) waves of equal size. (Mackenzie.)

Venous Pulsation in Children.—Pulsation of the jugular veins is not a very uncommon phenomenon in children. In conditions of de-

bility, and when there are symptoms of cardiac failure, it is not unusual to find flickering movements in the external jugular veins. Tracings from these movements cannot easily be obtained, but when there is pulsation of the internal jugular veins the wave is of sufficient magnitude to be graphically recorded. The auricular type of venous pulse occurs in children more frequently than is generally supposed, and it may exist whenever the right heart has become dilated from any cause. It is also sometimes a

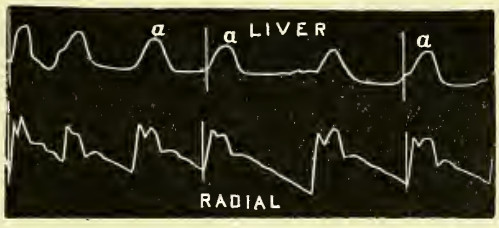


FIG. 136.—Simultaneous tracings of the liver and radial pulses. The liver pulse shows only one wave, the ventricular (*a*). The auricle is paralysed. (Mackenzie.)

symptom of tricuspid regurgitation, and the writer recently obtained excellent tracings from the jugular vein of a child of eleven years suffering from mitral disease, and where a distinct systolic murmur was present over the tricuspid area. The ventricular type of venous pulse also occurs in children. In three cases of grave cardiac disease tracings were obtained simultaneously with the radial pulse. All the patients were boys who suffered from very advanced mitral disease, accompanied with extreme hypertrophy and dilatation of the heart. The venous tracing in Fig. 137 is of the ventricular type, but a pulsation of this nature must be seldom obtained at such an early age. The patient was under the care of Dr. John Thomson,

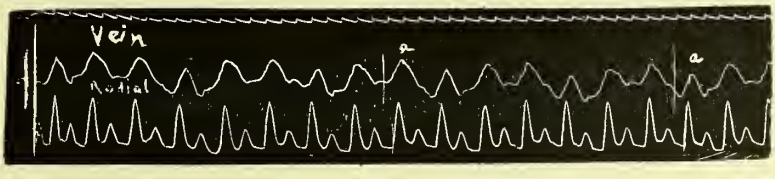


FIG. 137.—Venous pulse of the ventricular type in a boy æt. 9 years, suffering from advanced mitral disease with great dilatation and hypertrophy of the heart.

who kindly permitted me to record the movement. After some weeks of treatment the boy was in a greatly improved condition, but at the time when the tracing was obtained his right auricle must have been temporarily paralysed.

Pulses. *See* DIET (*Vegetable Food, Cereals, Pulses*).

Pulsus.—The pulse, especially in such expressions as *pulsus celer* (quick pulse), *pulsus bigeminus* (paired pulse), *pulsus paradoxus, pulsus plenus, pulsus tardus* (slow pulse), etc. *See* PULSE.

Pulveres.—Powders (*q.v.*). *See also* PRESCRIBING.

Pulvinar. — A cushion-like projection (Lat. *pulvinar*, a couch) on the posterior part of the optic thalamus of the brain.

Pump, Breast. *See* PUERPERIUM, PHYSIOLOGY; PUERPERIUM, PATHOLOGY (*Affections of Breasts and Nipples*).

Pump, Stomach. *See* TOXICOLOGY (*General Treatment of Poisoning*).

Punctum. — A point (plural, *puncta*), *e.g.* the *puncta lacrimalia* (openings into the lachrymal sac). *See* LACRIMAL APPARATUS, DISEASES OF (*Anatomy*).

Puncture. *See* CURETTAGE (*Dangers, Uterine Puncture*); ECTOPIC GESTATION (*Treatment, Puncture of Fœtal Sac*); HYDROCEPHALUS (*Treatment, Surgical, Puncture of Skull*); MORPHINOMANIA (*Diagnosis, Puncture-marks*); OVARIES, DISEASES OF (*Ovarian Cysts, Treatment by Puncture*); SPINA BIFIDA (*Treatment*).

Puncture, Lumbar. *See* SPINE, SURGICAL AFFECTIONS OF (*Lumbar Puncture*). *See also* HYDROCEPHALUS (*Surgical Treatment, Lumbar Puncture*); MENINGITIS, TUBERCULOUS AND POSTERIOR BASIC (*Diagnosis and Treatment, Lumbar Puncture*); MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Diagnosis*).

Pupil, The.

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Nervous Mechanism 435
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See also ABDOMINAL ANEURYSM (*Symptoms, Inequality of Pupils*); AORTA, ANEURYSM OF (*Symptoms, Pupils*); BRACHIAL PLEXUS, SURGICAL AFFECTIONS OF (*Tears and Contusions, Symptoms*); BRAIN, PHYSIOLOGY OF (*Third Nerve*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Anæmia, Symptoms*); BRAIN, SURGERY OF (*Concussion, Symptoms*); BRAIN, SURGERY OF (*Leptomeningitis, Symptoms*); BRAIN, CEREBELLUM, AFFECTIONS OF (*Hæmorrhage, Symptomatology*); BROMISM (*Symptoms, Pupils*); CATARACT (*Diagnosis*); COCAINE (*Physiological Action*); EYE, CLINICAL EXAMINATION OF (*Anterior Chamber, Iris, and Pupil*); GENERAL PARALYSIS (*Symptoms*); GLAUCOMA (*Causes and Pathology*); IRIS AND CILIARY BODIES; MEDIASTINUM (*Tumours, Symptoms*); MENINGITIS, TUBERCULOUS (*Symptoms*); NEURASTHENIA (*Symptoms, Pupils*); PHYSIOLOGY, NEURO-MUSCULAR MECHANISM (*Anatomy of Eye*, etc.); RETINA AND OPTIC NERVE (*Atrophy of Optic Nerve, Symp-*

toms); Sclerotic, Diseases of (*Sclerotitis, Pupil*); Spine, Surgical Affections of (*Injuries, Localising Symptoms*); Syringomyelia (*Symptoms*); Tabes Dorsalis (*Symptoms, Pupil Phenomena*); Uræmia (*Symptoms*); Vision, Field of (*Hemianopia, Hemiopic Pupil*).

It is remarkable how frequently help of the most important kind in solving the medical problems that are from time to time presented to us is afforded by a systematic and habitual close observation of the eye. Clues are indeed frequently obtained by this method, which becomes a second nature to one who has had training in ophthalmic practice, of instantly appreciating the general condition of the eye movements as the patient sits before him, their relative position, lid movements, and pupil appearances—clues which often lead directly to a correct diagnosis of cases of nervous disease which might otherwise have been entirely overlooked. Perhaps the most obvious disease which, by means of the eye symptoms it presents, can be diagnosed frequently the moment the patient enters the room, is Graves' disease, in which the bulging eyes, widely open and showing white sclera above the cornea, with dilated pupils and general aspect suggestive of intense terror, form a picture familiar to every student of medicine. Yet in cases of this disease in which all the usual ocular symptoms are absent, with the goitrous swelling carefully hidden from view by frilling or a high collar in the sex which affords the large majority of these patients, how frequently the tachycardia and the nervousness that is only too apparent are wrongly ascribed to hysteria or excitement!

The movements of the pupil are governed chiefly by two antagonistic muscles—the sphincter pupillæ, innervated by the third nerve; and the dilator pupillæ, which receives its nerve-supply through the cervical sympathetic. In addition to these influences, the size of the pupil will vary with the amount of blood in the vessels contained in the iris, and small oscillations of the pupil occur with each heart-beat. Thus congestion of the iris in iritis causes contraction of the pupil, while dilatation of the pupil is seen in chlorosis and other anæmias. In these latter, however, the dilatation is not alone dependent upon the condition of oligæmia, which is indeed not always present, but partly upon the stimulating effect of the venosity of the blood upon the pupil-dilating centre in the floor of the aqueduct. This effect is well seen in the rhythmic dilatation and contraction of the pupils which occur in Cheyne-Stokes breathing, either of cardiac or cerebral origin; dilatation of the pupil occurring at the beginning of the period of dyspnœa from simultaneous stimulation of the pupil-dilating and respiratory centres by the venous blood, the pupil contracting again towards the end of the dyspnœic period.

The *size of the pupils* varies with structural defects of the eyes, with the age and complexion of the person. In hypermetropia the pupils are said to be generally smaller than in myopia, owing to the constant tonic contraction of the ciliary muscle in the former, with the action of which contraction of the pupils is generally associated. Convergence is, however, more intimately associated with pupil contraction than is accommodation, but the centres for the three actions lie close together in the anterior portion of the third nucleus, and isolated paralysis of these three movements has been recorded. Habitual contraction may lead to permanent narrowing of the pupil, as is seen in engravers and etchers, jewellers, etc. If the act of convergence be produced without any accompanying contraction of the ciliary muscle, as can be done by placing two prisms, base outwards, in front of the eyes, and the gaze directed to a distant object, contraction of the pupils is seen to accompany the effort of convergence which is necessary to overcome the diplopia; whereas, conversely, if when the gaze is directed at a distant object two concave lenses are placed before the eyes, although accommodation is necessary to focus the object, yet the pupils do not contract.

In old age and in infancy the pupils are usually considerably smaller than in youth; and in light-complexioned persons with blue irides the deficiency of pigment, by allowing more light to pass through and stimulate the retina, causes the pupil to be generally smaller than in people of darker complexion. Many diseases of the eye are associated with alteration of the size of the pupil and with the pupil-reflex to light, such as vitreous opacities, choroiditis, retinitis, optic neuritis, and atrophy, the pupil becoming dilated and losing its contractility to light roughly in proportion to the degree of loss of vision. In many persons, however, who have no perception of light remaining after retinal disease or optic atrophy, the pupil may still be observed to contract slightly under the stimulus of a strong light, owing to the fact that there are special fibres contained in the optic nerve, much smaller than the visual fibres, which are concerned in the conduction of the impulses for the light reflex.

In acute glaucoma the pupil is dilated, often oval vertically, and the light reflex may also be lost from the excessive intraocular pressure rendering the iris anæmic, and paralysing the nerve-endings in the sphincter pupillæ. *Many toxic influences affect the pupil.* In the algid stage of cholera the pupils may be widely dilated, whereas in typhus the pupils are generally very small. Numerous drugs also have power over the pupil either by direct action on the nerve-endings in the iris, or by affecting the pupil-contracting or pupil-dilating centres in the pons. Cocaine applied to the

eye stimulates the endings of the sympathetic, and also contracts the vessels, thus producing dilatation of the pupil. Atropine also stimulates the dilator pupillæ, but also paralyses the endings of the third nerve in the sphincter pupillæ, causing the most extreme form of mydriasis. Eserine and pilocarpine, on the other hand, produce myosis by stimulation of the endings of the third nerve. In post-diphtheritic and alcoholic neuritis the pupils may be somewhat sluggish to light.

Opium, again, causes contraction of the pupil in most stages of its action, and chronic opium-takers may sometimes be detected by means of their pin-point pupils. Chloroform anæsthesia also causes contraction of the pupils in proportion to the degree of anæsthesia up to a certain point, beyond which rapid dilatation occurs from paralysis of the pupil-contracting centre. Deep sleep similarly produces contracted pupils by means of cessation of tonic action of the pupil-dilating centre. Psychical states in general react on the pupil through its centres in the pons, and the pupil may be seen to vary rapidly in size in different stages of emotion, anger contracting the pupil, and love causing dilatation, according to Sir Samuel Wilks. In proof of the effect of anger in contracting the pupil he quotes the contraction of the pupils that occurs in a parrot which has been annoyed. This case is, however, not comparable to that of man, for the parrot has voluntary power over the contraction and dilatation of the iris, and atropine has no effect in dilating its pupil. In hysteria and neurasthenia the pupils may be slightly unequal, though some inequality is often physiological, and in hysteroid states oscillations of the pupil, known as hippus, may be met with. This condition may also be met with in epileptics. A certain amount of rhythmic oscillation of the pupil is normal after exposure to light, strong contraction at first being succeeded by slight dilatation, the size of the pupil oscillating until it comes finally to rest in the contracted state. This is produced by the initial contraction shutting out a large proportion of the light entering the eye, and thus diminishing the stimulus; hence the succeeding dilatation, which again lets in more light, which again causes contraction. Occasionally a condition known as the paradoxical pupil reaction is seen, in which, under the stimulus of a strong light, though the pupil at first contracts, the succeeding oscillations are followed by considerable dilatation, which persists in spite of the continuance of the stimulus of the light falling on the retina. This paradoxical pupil reaction may be met with in the early stages of general paralysis of the insane, and, if well marked, will be therefore of bad prognosis.

In the elucidation of nervous diseases the condition of the pupil is often of the utmost importance, and it is therefore necessary to have a knowledge of the minute anatomy of the pupil-reflex arcs for contraction and dilatation, as well as of these centres. The light reflex, starting from the retina, passes up special fine fibres in the optic nerve, which undergo a semi-decussation at the chiasma like the visual fibres, passes up the optic tract of each side, and leaves the tract just anterior to the external geniculate body, passing by the anterior brachium to the grey matter of the anterior corpus quadrigeminum, where the fibres must make connection with nerve-cells, since ascending degeneration resulting from lesion of the optic tract cannot be traced farther than the grey matter on the surface of the corpus quadrigeminum. From this point to the third nerve-nucleus lying beneath the floor of the anterior end of the aqueduct its course is not certainly known, but in all probability it passes by means of Meynert's fountain decussation to the third nuclei of the same and of the opposite sides. The anterior end of the nucleus is specialised as the sphincter pupillæ centre, and it is generally supposed that the centres of the two sides are united by commissural fibres. From the nucleus the efferent part of the reflex arc is the third nerve, ciliary ganglion, and short ciliary nerves to the iris. Lesions of any portion of this reflex arc will have the effect of abolishing the pupil reflex, whether the endings of the third nerves are paralysed by atropine or in disease of the optic nerve. Owing, however, to the semi-decussation of the fibres at the optic chiasma, and perhaps also to the commissural union of the centres in the third nucleus, there is a consensual reaction of both pupils when only one is exposed to light. By means of testing both the direct and consensual reflex to light in each pupil separately, much information may be gained both as to the site of a lesion causing blindness, and of one producing loss of the light reflex. For example, in optic atrophy of one eye following gunshot injury of the orbit, light thrown on the blind eye will not produce contraction of either pupil, but light thrown on the good eye will cause brisk and equal contraction of both pupils, since the stimulus can pass up both optic tracts, and both third nuclei are affected. Both pupils will also contract equally on convergence. In such a case there is therefore loss of consensual pupil reflex to light in the good eye, whereas the consensual reflex is unimpaired in the blind eye. In such a case the injury must lie in the afferent portion of the reflex arc, and further, it must lie between the retina and the optic chiasma, since a lesion of the optic tract does not abolish the direct pupil reflex in one eye completely, but will affect the corresponding halves of both retinæ, producing the phenomenon known as the hemiopic pupil of Wernicke. When light is focussed on the anæsthetic half of either retina no pupil reaction

results, but when focussed on either seeing half consensual pupil reaction in both eyes is observed. Knies has also theorised that this hemiopic pupil reaction must be obtainable in cases of lesion of the reflex fibres after they have left the optic tracts on their way to the third nerve nuclei, without any corresponding hemianopic loss of vision. This symptom has not been actually recorded, but might conceivably be met with in lesions of either anterior corpus quadrigeminum or of its brachium. Any lesion damaging the visual path above the external geniculate body, whether in the optic radiations or in the cortex of the occipital lobe, will produce hemianopia without interfering with the pupil reflex, and the pupils will react normally to light even though the lesion be bilateral, producing total blindness.

The reflex arc for the sympathetic control over the dilating mechanism of the pupil is considerably more complex than that of the light reflex. The nuclear centre lies close to the third nucleus in the floor of the aqueduct, and external to it. Afferent impressions reach this centre along numerous paths; indeed, strong sensory impressions from almost any part of the body may result in stimulation of this centre. A usual way of testing the integrity of this reflex is to pinch the skin of the neck sharply, when dilatation of the pupil results. Equally potent, however, is a pin-prick on the inside of the nose, when the fifth nerve must serve as the afferent loop of the reflex arc. Intestinal disorders, such as worms and painful colic, are also said to react on the pupil in the same way. Moreover, impulses passing downwards from the cortex are continually affecting this centre, as is seen in the continual alteration in size of the pupil under emotion, irrespective of alteration in the intensity of light. It is possible, as some think, that there is a special centre for regulation of the size of the pupil in the neighbourhood of the head and eye centre in the posterior portion of the frontal lobe. Haab has described an "attention reflex" in which contraction or dilatation of the pupil occurs according as the attention is concentrated on a bright or dark object respectively on either side, while the gaze is fixed on a medium-coloured wall in front. With this may be compared the dilatation of the pupil which occurs on concentrating the thoughts on a long-past event, and contraction on thinking of the distant future—a common "thought-reading" experiment. Gifford and Galassi described pupillary contraction on forced closure of the lids. Piltz finds this "orbicularis reflex" consensual, and present even in blind eyes and in cases with Argyll-Robertson pupil. The efferent path of the sympathetic impulses to the pupil is more definitely known; starting from the centre in the floor of the aqueduct, the fibres traverse the pons and bulb with the ascending root of the fifth nerve, and pass down the spinal cord in the lateral columns of the same side as far as the second dorsal root. They leave the cord at this level by the anterior root, pass by the rami communicantes to the inferior cervical ganglion, thence along the cervical sympathetic to the superior-cervical ganglion, whence they pass along the internal carotid artery, and so enter the cranial cavity, and reach the Gasserian ganglion. Reaching in this manner the fifth nerve, they accompany the ophthalmic division of this nerve, its nasal branch, and thence reach the ciliary ganglion, reaching the iris eventually by the long ciliary nerves. Lesions of the pons, bulb, and especially of the lateral columns of the cervical cord, as well as of the inferior cervical ganglion and cervical sympathetic, such as thoracic aneurysm, mediastinal tumour, or enlarged glands in the neck, will produce paralysis of the dilator pupillæ and contracted pupil. Such contracted pupils do not dilate in shade, and the condition is therefore more obvious when both eyes are shaded from the light. They still contract slightly to light, both directly and consensually, but not quite so briskly as a normal pupil. Indeed, the reaction to light in this contracted pupil may be so sluggish as closely to resemble the Argyll-Robertson pupil. There is also present slight ptosis from paralysis of Müller's muscle, and the eye will appear slightly sunken. Anterior poliomyelitis, posterior sclerosis, and sclerosis of the crossed pyramidal tracts in the cervical region, do not produce sympathetic paralysis of the pupil, but gross lesions of the cord, which affect also the lateral column, as cervical myelitis, syringomyelia, and fracture-dislocations of the cervical spine will be accompanied by this symptom. This condition of sympathetic ptosis and contracted pupil will often afford the first clue in the diagnosis of syringomyelia from progressive muscular atrophy. Irritant lesions of the spinal cord in the lower cervical region at first cause dilatation of the pupil, later contraction from paralysis of the sympathetic fibres. Schiff described a paradoxical reaction, the pupil contracting instead of dilating on faradisation of the sympathetic; this I have seen in tabes. Dilatation of the pupil is said to occur in the early stages of aneurysm of the second part of the arch of the aorta. This I have never met with, the common condition being contracted pupil with slight ptosis from paralysis. Slight optic neuritis not infrequently accompanies sympathetic ptosis resulting from recent lesions of the cervical cord.

Since cocaine dilates the pupil by stimulation of the peripheral endings of the sympathetic in the iris, this fact has been taken advantage of in the differential diagnosis of complete and partial transverse lesions of the cervical cord. If the pupil will no longer dilate to cocaine, the inference is drawn that the sympathetic fibres are completely degenerated, and that therefore

the cervical lesion is practically completely transverse. This symptom must, however, accord with the other facts bearing on this point to be of value; for example, if in a case of fracture-dislocation of the sixth cervical spine there is complete motor paralysis below the lesion, and a fortnight later no dilatation of the pupils to cocaine, the inference would be that the lesion was completely transverse, if there were also absolute anæsthesia below the first intercostal space; yet if the patient could definitely feel a pin-prick on his foot or leg, it is obvious that the lesion cannot be completely transverse.

A most important symptom to recognise is that known as the *Argyll-Robertson pupil.* This was first described in the small pupils, or spinal myosis occurring in tabes, in which it was noticed that the pupils contracted normally to accommodation and convergence, but not to light, although vision might be perfectly good. The essential point, however, is the loss of the pupillary reflex to light in cases in which it can be demonstrated that the pupil is not fixed from iritic adhesions or other intra-ocular cause, and in which the afferent portion of the reflex in the optic nerve is uninjured, as proved by the vision being still preserved. The pupils are generally small, sometimes minute, so-called pin-point pupils, and there is then also sympathetic paralysis present, with loss of reflex dilatation from stimulation of the skin. Sometimes the pupils are unequal, in which case the loss of the light reflex is mostly more advanced on the side of the smaller pupil. Rarely, the phenomenon is uniocular, the other pupil remaining normal for a time, though in the majority of these cases both pupils are ultimately affected. In other cases, again, the pupils may be medium dilated, and occasionally widely dilated; there is then generally no contraction on convergence or accommodation. This symptom is found in the very large majority of cases of tabes, and in the later stages of general paralysis of the insane, in which the pupils may also be irregular in outline. It may be occasionally met with in other nervous diseases; I have met with it in progressive muscular atrophy, in lead palsy, in aortic aneurysm, and occasionally in patients presenting themselves for slight ailments, in whom no other sign suggestive of tabes or general paralysis could be found, but who had suffered previously from syphilis. As Gowers first pointed out, the true significance of the symptom lies in its indicating, with almost absolute certainty, previous syphilis. It is a sign of nerve degeneration resulting from the late effects of the poison, and is therefore by far the most commonly met with in tabes and in general paralysis, conditions of nerve degeneration dependent in great degree if not entirely on previous syphilis. Congenital syphilis may occasionally give rise to juvenile tabes or general paralysis, when this symptom will almost certainly be present. Argyll-Robertson pupil is perhaps the most constant symptom in tabes, and is probably the earliest recognisable symptom, being often well marked in cases of tabes in which the knee-jerks may be still quite lively. Cases of tabes in which the pupil reaction is perfect are most exceptional, and the diagnosis should always be most carefully considered in such cases. Broadly speaking, the observance of this symptom will lead to the presumption that the case is probably one of tabes, or possibly general paralysis. If loss of knee-jerk be present in addition, the diagnosis of tabes will be confirmed. It is often quite easy to recognise the presence of this symptom almost at the first glance at a patient, inequality or immobility of the pupils, especially when facing the window, giving the clue, and leading to the actual test being made. In making the test, it is better to expose the eyes to strong diffused daylight than to light focussed from a lamp, the general stimulation of the retina being better from daylight, and there is then less chance of the pupils contracting with associated accommodation. Both pupils should be shaded together, and exposed to the light at the same time, in order to obtain the maximum of stimulus, and at the same time to compare the comparative briskness of contraction on the two sides, since marked diminution in briskness of contraction will be almost as significant as the fully developed symptom. The anatomical lesion underlying this loss of the light reflex has not yet been demonstrated, but presumably it is due to sclerosis of the pupil reflex fibres between the point at which they leave the optic tract and the sphincter centre in the third nucleus, perhaps in Meynert's decussation. It is thought by some that it is due to nuclear degeneration, and this must almost certainly be present when there is co-existent loss of contraction to accommodation. In most cases, however, the pupils contract briskly on convergence, and it is difficult to conceive how the sphincter pupillæ centre could be paralysed for the light reflex only, and yet work normally for convergence. We may perhaps assume that there is sclerosis of the endings of the fibres in Meynert's decussation where they impinge on the third nucleus, and that in some cases the degenerative process extends into the nucleus itself.

Alteration in the pupils is often to be observed in *acute cerebral disturbances.* At the commencement of an epileptic fit both pupils become contracted, an irritative phenomenon, while in the transient stage of flaccid paralysis, after the convulsions have ceased, the pupils are widely dilated and insensitive to light. In meningitis the pupils are contracted in the first stage of cerebral irritation, unequal in the next stage of compression, and dilated in the third stage of paralysis. In acute mania and delirium the

pupils are dilated, and often unequal or sluggish to light in alcoholic insanity. Cerebral hæmorrhage may produce inequality of the pupils, that on the side of the hæmorrhage being more often slightly dilated, especially if the bleeding has extended into the lateral and third ventricles. Hæmorrhage in the pons is frequently accompanied by minute contraction of the pupils, from irritation of the third nuclei, but this is not invariably present, and it is difficult to draw any precise deductions from the condition of the pupils in cerebral hæmorrhage, so variable are the effects. In cerebral tumour no alteration in the pupil will be seen unless there is pressure on the third nerve, when moderate dilatation and loss of reflex action will accompany the ptosis and divergent strabismus characteristic of paralysis of this nerve. Third nerve palsy from either nerve or nuclear lesion is common in tabes, and may be either partial or complete. When the ptosis and muscular impairment are only slight, the pupil reaction to light should not be completely lost, but if this is so, it will indicate the additional presence of the Argyll-Robertson pupil, which will often confirm an otherwise doubtful diagnosis of tabes in a case presenting itself for an incomplete paralysis of the third nerve. Fifth nerve palsy might be expected to be frequently associated with small pupil on the same side from involvement of the sympathetic fibres passing over the Gasserian ganglion, yet this is quite the exception, and I have only seen this association twice.

Pupillary Membrane. — A transparent, vascular membrane which closes the pupil in early fœtal life; it may remain in part as a malformation in the form of fibres, tags, or loops (*persistent pupillary membrane*). *See* Iris and Ciliary Bodies (*Congenital Abnormalities of the Iris*).

Pupiloscopy. *See* Retinoscopy.

Purgatives. *See* Cathartics; Constipation; Pharmacology; etc.

Purgen.—An aperient drug, being chemically phenolphthalein, given in doses of from 1 to 5 grains; aperione. *See* Prescribing.

Purification. *See* Ventilation and Warming (*Purification of Air*); Water (*Purification*).

Purin Bodies. *See* Physiology, Excretion (*Urine, Non-Urea Nitrogen, Diureides*). *See also* Liver, Physiology of (*Regulation of Supply of Proteids, Uric Acid, Xanthin*, etc.); Uric Acid (*Chemical Characters, Purin Ring*).

Purkinje's Cells. *See* Physiology, Neuro-muscular Mechanism (*Cerebellum, Structure*).

Purkinje's Fibres.—Peculiar muscular fibres showing incomplete striation, found in the sub-endocardial tissue of the heart in some of the lower animals, in the human subject in early infancy and occasionally in the adult.

Purkinje's Figures or Images. *See* Physiology, Neuro-muscular Mechanism (*Vision, Stimulation of Retina*).

Puroform.—An antiseptic said to consist of zinc and formic aldehyde with thymol, menthol, and eucalyptol.

Purple, Visual. — Rhodopsin. *See* Colour Vision (*Physiological Considerations*).

Purpura.

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Definition.—The essential point in purpura is the occurrence of extravasations of blood into the skin, which are spontaneous and not due to injury. Strictly speaking, purpura is in all cases merely a symptom. In many instances the disease of which it is a symptom is fairly well known, and a great class of "symptomatic" purpura is therefore described. But in certain instances, more especially where hæmorrhage from mucous surfaces is associated with the cutaneous extravasation, the conditions upon which the hæmorrhage depends are unknown, and in default of such knowledge the term "purpura" is borrowed from the one striking symptom, and is used to designate "a disease" with the addition of various distinguishing adjectives.

Character of the Cutaneous Hæmorrhage.—Petechiæ form the chief feature in all kinds of purpura. They are red or purple spots which assume a rusty colour as they disappear. They commonly appear in successive crops. They may be thickly distributed over the whole surface of the body, and in fatal cases the serous and mucous membranes are generally found to be similarly affected. In size they vary from a mere speck to a spot half an inch in diameter. The smallest size is often seen as the result of flea-bites. In shape they are rounded, irregular, or star-shaped. They do not fade on pressure. Except in scurvy they have no relation to the hair-follicles. After their appearance they do not increase in size, except by confluence with neighbouring petechiæ. As a rule they are not raised above the level of the skin, but sometimes the centre is elevated into a papule. Occasionally some hæmorrhages are in the form of streaks (vibices). In association with petechiæ, purplish bruises (ecchymoses) are often seen, sometimes measuring several inches in length. As these fade they show the brown and greenish yellow tints which are familiar in the traumatic bruise. Occasionally also large extravasations in the subcutaneous tissue are met with (hæmatomata).

Etiology and Pathology.—From a consideration of the conditions under which a "symptomatic purpura" arises, we are justified in concluding that a purpuric eruption (excluding that of hæmophilia) may be produced: (1) by the presence in the blood either of bacteria themselves or of their poisonous products; (2) by the presence in the blood of some poison of non-bacterial origin, either introduced from without or manufactured in the body; (3) by some alteration in the formed or other constituents of the blood; (4) by vaso-motor influence. All instances of symptomatic purpura may be grouped in these four classes, which may be termed infectious, toxic, cachectic, and nervous respectively.

Although a bacillus such as that of plague, a toxin such as that produced by the bacillus diphtheriæ, a drug such as iodide of potassium, or a high degree of anæmia may be spoken of as the cause of hæmorrhage, we are very far from understanding the exact method by which rupture of the vessel wall is effected. Doubtless some widespread deleterious influence is exerted by these agents upon the vessel. But in the production of petechiæ something more than this is needed. There must be some subsidiary local factors which determine the site of the hæmorrhage. Slight injury, sudden local alterations in blood pressure, capillary thrombosis, the unsupported state of a vessel, and the influence of gravity may conceivably contribute something towards the production of petechiæ. For example, in any purpuric state the prick of a needle will produce a hæmorrhagic spot. Gravity certainly plays a small part in certain cases of recurring arthritic purpura. Moreover, it has been shown by Unna that the point at which the cutaneous vessel ruptures is its point of least resistance. Rupture occurs in the lower part of the cutis, where the vessels lose their adventitia and media, and practically become capillaries without obtaining the support of the rigid cutis.

Various changes have been described in the vessel wall, such as endarteritis, hyaline and amyloid degeneration. It is more than doubtful, however, whether such changes would be favourable to bleeding. At any rate, in view of the clinical history of the sudden appearance of petechiæ in previously healthy individuals, such an explanation is now generally discarded.

Histological evidence as to the local cause of petechiæ is scanty. Thrombosis of small veins has been observed, but considering the freedom of anastomosis of cutaneous vessels, this cannot be regarded as important. Capillary emboli have been found in purpura dependent upon generalised sarcoma. A more promising explanation lies in the bacterial emboli, which, with or without capillary thrombosis, have been found in many cases of purpuric eruption appearing in infectious and septic diseases. On the other hand, while careful search has failed to reveal such an appearance in the hæmorrhagic spots of variola and diphtheria, the capillaries in the papillæ of the skin in scarlet fever have been found plugged with cocci without the occurrence of any hæmorrhage. The evidence is not strong enough to bear a conclusion.

The conditions, however, under which a symptomatic purpura occurs are known, and have been already enumerated. We may go so far as to suppose: (1) that in all of them, perhaps even in the unimportant nervous class, a circulating poison or an altered blood-state sets up in small vessels and capillaries a preparatory change in the direction of impaired vitality and loss of cell-cohesion, the exact nature of the change being unknown; and (2) that in this state various local factors determine rupture and extravasation of blood.

Our knowledge of the origin of "purpura as a symptom" is thus imperfect. When, however, we consider the nature of "purpura as a disease" we are reduced to pure hypothesis. The age-incidence of all forms of the disease is the only well-defined fact, the liability of children and young adults being very prominent. Both sexes are affected, and no great difference in their liability is noted. The family history throws no light on the etiology, and no clue is gained from the previous history or surroundings of the patient. A review of a large number of cases leads to the provisional recognition of five forms of the disease, which are named simple, Werlhof's, arthritic, Henoch's, and febrile purpura respectively. It must be re-

membered that this classification is the outcome of purely bedside study. These forms may prove to be degrees or varieties of one disease, or they may be shown to be as distinct one from another as typhoid from typhus. To decide the question of their relationship there is needed a far deeper knowledge of their bacterial and chemical conditions than we now possess.

It will be noted that these five forms have many important points in common, each one, however, possessing some characteristic feature. In (1) simple purpura the hæmorrhagic tendency is almost confined to the skin, and the illness is mild and ends in recovery. In (2) Werlhof's purpura all mucous membranes and many viscera share in the tendency, and the illness becomes more severe, is often of longer duration, and may end fatally. In (3) arthritic purpura, joint-pains are added to the hæmorrhagic element, and a proneness to recurrence becomes a prominent feature. In (4) Henoch's purpura severe colic and visceral hæmorrhage are additional symptoms. In (5) febrile purpura we have a well-defined severe disease approaching closely in type to a specific fever such as smallpox, with pyrexia and a tendency to necrosis of certain tissues.

It is true that examples may be picked out from these various forms which, placed side by side, will present strongly differentiating features. But it is equally true that, if a large series of cases is studied, instances are met with which seem to lie as links between them. At any rate, on the ground of clinical affinity, we are justified in looking upon simple purpura and Werlhof's purpura on the one hand, and arthritic and Henoch's purpura on the other hand, as forming two large groups, while the febrile form lies somewhat apart from them both.

As regards their causation there is abundant room for speculation. Nothing certain is known, but their close analogy with such instances as iodic purpura (toxic) and diphtherial purpura (infectious) compels us to regard them as coming under one or other of these two heads. Febrile purpura bears such a close resemblance to a specific fever that many attempts have been made to prove its bacterial origin. Their failure need not militate against the view. Bacteria of many kinds have been found in the blood and tissues, but the observations are vitiated by the fact that they were made some time after death. Their importance is not necessarily increased by the fact that the injection of cultures of these organisms has produced hæmorrhagic septicæmia in animals. Among these microbes are streptococcus, staphylococcus, a capsulated bacillus belonging to the colon group, and other bacilli not classified. The blood, if taken direct from a vein during life, is generally sterile. Both streptococcus and staphylococcus have, however, been grown from it; but, considering the common existence of a sloughing process in the mouth or throat or elsewhere in these cases, their presence cannot be regarded as necessarily meaning more than the supervention of a septicæmia. The common failure to find micro-organisms in the blood is not surprising, if we consider that the specific organism of so many of the recognised specific fevers is still unknown.

A. Symptomatic Purpura

Excluding hæmophilia, the conditions of which purpura is a frequent or occasional symptom may be grouped in four classes. For the most part the form of hæmorrhage is the petechial eruption.

(1) *Infectious.*—Nearly all the specific fevers may be attended by a purpuric eruption, especially smallpox, diphtheria, measles, scarlet fever, and cerebro-spinal fever. Such an eruption betokens unusual severity in the disease. In typhus the rash is always of this nature. In typhoid fever petechiæ and vibices have been rarely seen. Petechiæ are not uncommon in yellow fever and in plague. They occur in malignant endocarditis, pyæmia, and septicæmia; more rarely in syphilis, gonorrhœa, malaria, pneumonia, and erysipelas.

(2) *Toxic.*—Snake-poison produces large extravasations of blood into the skin, as well as hæmorrhage from mucous surfaces. A copious petechial eruption may follow the use of diphtheria antitoxin A large number of drugs can produce petechiæ. Phosphorus, iodide of potassium, chloral, arsenic, quinine, mercury, salicylic acid, copaiba, ergot, belladonna, antipyrin, and opium and its alkaloids, may be included under this head. In phosphorus-poisoning hæmorrhage often occurs into the skin as well as from mucous surfaces. Iodide of potassium will produce in a few exceptional individuals an attack which closely resembles purpura simplex. As regards the other drugs enumerated such an effect is exceedingly rare. Petechiæ are also not uncommon in certain ill-defined states of poisoning, such as arise in nephritis, cirrhosis of the liver, and malignant jaundice.

(3) *Cachectic.*—A purpuric eruption is occasionally met with in cases of generalised sarcoma. A few petechiæ are not uncommonly seen on the lower extremities in cases of advanced phthisis or carcinoma, and in fact occasionally just before death from any form of long-standing disease. They may appear also in extreme old age. They are common in leucocythæmia, pernicious anæmia, and lymphadenoma. In scurvy and in scurvy-rickets, petechiæ, ecchymoses, and large subcutaneous extravasations of blood are common phenomena.

(4) *Nervous.*—Petechiæ occasionally occur in locomotor ataxy, especially in association with

lightning pains. They have been seen also in myelitis, alcoholic neuritis, and neuralgia, and even in hysteria after severe emotion or over-excitement.

B. Purpura as a Disease

Symptoms and Course.—(1) *Simple Purpura.*—This form is almost confined to children of both sexes under the age of fifteen. The family history throws no light on its causation, but it is noteworthy that occasionally more than one child of a family is attacked. The surroundings of the child seem to play no part in the origin of the disease, for it frequently appears in children of good position, as to whose diet and life no fault can be found. In most cases the child appears to be healthy and well-nourished up to the onset of the symptoms. Sometimes it is ill-nourished or rickety. It may be noted that the attack is sometimes preceded or ushered in by diarrhœa, and occasionally indigestible or doubtful food, such as sausages, has produced gastric disturbance a day or two before the onset. Herpes has been known to appear on the lips at the beginning of an attack.

As a rule, without any appearance of ill-health, the first thing which attracts attention is the appearance of petechiæ. They may be few at first, perhaps appearing in the first place about the neck as often as anywhere, but successive crops are added, until nearly the whole surface of the body is thickly studded with red, purple, or brown spots, having the characters already described. Large patches are sometimes formed by confluence of petechiæ. The face may be affected as well as the trunk and extremities, but the palms of the hands and the soles of the feet usually remain free. A few petechiæ may often be seen inside the lips and on the palate and tongue. They rarely if ever occur on the conjunctivæ or retinæ. In very slight cases they may be confined to the lower extremities. Rarely some red wheals are present among the petechiæ. Ecchymoses are common, and are readily produced by any rough handling. Hæmatomata occasionally develop, especially about the buttocks, thighs, and calves, and a bluish-black brawny mass as large as a man's hand is sometimes met with in these situations. On such a mass a bulla containing sanguineous fluid may be formed, but this appearance is not so common here as in the febrile form of purpura. Close observation will sometimes detect a little œdema of the backs of hands and feet. In a few cases there may be some slight epistaxis at the beginning of an attack, or slight oozing of blood from the gums, but with these exceptions the hæmorrhage is confined to the skin. Occasionally there is evidence of slight pains in the joints.

In all cases the general condition of the child is in striking contrast with the cutaneous appearance. Often there is no indication or feeling of ill-health whatever, and all functions are naturally performed. Sometimes there is loss of appetite and a little restlessness, and perhaps for a few days the temperature may reach 100° in the evening. The child is, however, often pale when it comes under observation, and sometimes the anæmia increases during the illness, the red corpuscles falling as low as 3,700,000 per c.mm. The leucocytes show no change in number or quality. There is no affection of the joints, no morbid appearance about the tonsils, and no evidence of intestinal disturbance beyond the occasional initial diarrhœa already mentioned.

Recovery is rapid and uniform, the length of the illness seldom exceeding a fortnight. No tendency to recurrence can be traced.

(2) *Werlhof's Purpura.*—This form, if we adhere to Werlhof's description of it, written in the eighteenth century, is a rare disease. It is clear, however, that his description must be amended in two important particulars. But though more cases are thus brought under the term, it still remains comparatively uncommon.

All that has been said of purpura simplex concerning the age and previous history of the patient, and the mode of onset and the early symptoms, holds good for this "morbus maculosus" of Werlhof. As a rule, it is not until a more extensive hæmorrhage has set in, and the illness assumes a severe aspect, that the diagnosis of the simple form is given up, and the more serious type of disease is recognised. The distinction between them is artificial and unnecessary.

The disease has therefore a gradual onset. For some days, or less commonly a week or two, crops of petechiæ continue to appear, perhaps the nose bleeds spontaneously on one or two occasions, and a little blood-clot may be seen between the teeth. The patient is anæmic, but as a rule presents no other evidence of illness. The hæmorrhagic tendency then rapidly increases. Epistaxis may be severe, and may necessitate plugging of the nostrils. The gums ooze freely, but if the adherent clot is removed they are seen to be pale and not swollen. Blood is vomited, and appears in the stools and in the urine. There may be expectoration of red blood, or of a blood-streaked sputum. Bleeding from the uterus and from the auditory meatus may occur. Hæmorrhages into the retina are not uncommon. Ecchymoses and hæmatomata may appear in various parts of the body. The tongue remains clean and moist, there is little or no pyrexia, and the appetite is commonly maintained. The blood shows the changes associated with loss of blood, and the red cells usually fall below 2,000,000, and sometimes below 1,000,000 per c.mm. The waxy pallor of the skin is striking, and the main complaint of the patient is the feeling of faintness.

According to Werlhof's description, the

hæmorrhagic tendency ceases at the end of five or ten days, and rapid recovery ensues without complication. Both these points must be modified in the light of wider experience. In the first place, the disease may run a much longer course. A patient may present the symptoms above described with hardly any intermission for at least two months, and with intermissions a case may occasionally run on for a year. In the second place, a fatal event is by no means uncommon, and the illness must always give rise to anxiety. By the continuance and profuseness of the hæmorrhage the anæmia reaches danger-point. The patient becomes, not restless, as is the case after a single large hæmorrhage, but apathetic and drowsy, and so dies exhausted; or at any point in the illness hæmorrhage into the brain, or upon its surface, may prove fatal.

In young children cases of great malignancy have been observed, and have been termed "purpura fulminans." With a sudden onset the cutaneous and visceral hæmorrhages are overwhelming, and death occurs in two or three days. In a recorded case, still more acute, no visceral hæmorrhage occurred, but the cutaneous hæmorrhage was extensive. Within a few hours all four limbs were swollen and discoloured by infiltrated blood, and the child died within twenty-four hours of the onset.

(3) *Arthritic Purpura.*—This form is a disease of adolescence and early adult life, and it is perhaps more common in males than females. The patient suffers for a few days from slight or severe pain in various joints, especially the knees, ankles, elbows, or wrists. Sometimes the joints are swollen and tender, but the pain may be severe, even when there is very little evidence of inflammation of the joint structures. Up to this point the condition will be regarded as a subacute rheumatism. Petechiæ now appear. As a rule, they are not distributed over the whole body, as in purpura simplex, though a few may be scattered over the trunk, and even on the mucous surface of the lips. They occur mostly on the lower extremities, especially on the shins and ankles, and they often show a tendency to be aggregated around an affected joint. Their distribution is symmetrical, as is the joint affection. In this form of purpura the centre of the hæmorrhagic spot is often elevated into a papule, which is commonly visible owing to its pallor. Sometimes in the centre of a spot may be seen and felt a pale spherical nodule, which is found on microscopical examination to consist of decolourised blood-clot. On the larger hæmorrhagic patches blebs filled with blood-stained serum may develop, and occasionally a little oozing will occur, so that a patch becomes surmounted with a crust of dried blood. Ecchymoses may often be observed, but in most cases the prevailing type is the simple petechial eruption. Slight œdema of feet and shins, or of backs of hands and forearms, is not uncommon. In some cases there is slight pyrexia, but often the temperature is normal throughout the illness. There is very little constitutional disturbance. Sweating is not a marked symptom, except in severe cases, and the skin is often dry and harsh.

The articular pains subside in a few days; the petechiæ, which appear in successive crops, become rusty and fade, and the patient may be well in a few weeks. But there is a strong tendency to recurrence. Though the patient remains in bed under treatment, the same phenomena, articular and cutaneous, are often repeated at an interval of a few days, and similar attacks, slight though they are, may carry the illness over many weeks. Even when the natural tendency to recurrence seems to have subsided, it will often be found that the first attempt to get the patient out of bed will result in another attack, and some months may elapse before the patient can pass the whole day in the erect attitude. In these later attacks the petechiæ are the marked feature. The articular pains may be very slight, often only stiffness is complained of, and as a rule there is no pyrexia.

The relation of this affection to acute rheumatism is uncertain. Strong arguments against any connection between the two conditions are the immense disparity in the frequency of their occurrence, and the moderate and often trifling nature of the joint affection. And as Scheby-Buch has pointed out, there is no form of purpura which does not occasionally present articular pains, and yet the term "rheumatic" can hardly be extended to all of them. Further, similar articular pains are often met with in cases of acute septicæmia, and do in fact sometimes lead at first to an erroneous diagnosis of rheumatism. Under the head of arthritic purpura must be provisionally included certain cases which are of less common occurrence, but of sharper onset and greater severity. As regards the condition of the joints and the general course of the illness, they do not require separate description. But they present some peculiar features. In the first place, wheals are conspicuous among the petechiæ, whence the name "purpura urticans" is derived. The second characteristic is the extensive œdema. The eyelids, cheeks, alæ nasi, lips, and chin may be enormously swollen and tense, studded with petechiæ or discoloured with ecchymoses. The hands and feet and the neighbourhood of any joint may present a similar appearance. The writer has seen the penis red and œdematous, with a wheal and abundant petechiæ on its surface. In these cases pyrexia is often more marked. The urine may contain blood or albumin. Rarely blood appears in the stools. Diarrhœa has occasionally been noted. It

should be noted that tonsillitis may usher in such an attack, and even when no throat symptoms are complained of the fauces are often found to be reddened. Endocarditis, pericarditis, and pleurisy have occasionally been noted; but in these cases there is a previous history of acute rheumatism.

(4) *Henoch's Purpura.*—This variety of purpura, which was described by Henoch in 1874, is most common in young people, the majority of cases occurring between the ages of seven and fifteen, and it is rare after thirty. Males seem to be more liable than females. Nothing is known as to its causation. It appears in children of good position, as well as in those who live under unhealthy conditions.

In its main features this form bears a close resemblance to arthritic purpura. The patient complains of vague aches and pains, such as occur at the onset of most specific fevers, with some feeling of illness. Soon one or more joints become painful, with little or no external evidence of inflammation. The appearance of slight œdema, the eruption of petechiæ, and sometimes of ecchymoses and wheals, and perhaps a slight rise of temperature, complete the resemblance to arthritic purpura.

After a few days, however, certain special symptoms are added. The child has paroxysms of severe abdominal pain, with obstinate vomiting and at first constipation, so that the picture of lead colic is recalled. But the vomit later is found to contain blood, diarrhœa generally sets in, the stools being black with altered blood; epistaxis is not uncommon, and there may be a little oozing from the gums. The pain is undoubtedly severe, the child cries aloud in the attacks, and there may be some collapse, the face becoming drawn and the pulse quick and small. The pain is generally referred to the umbilical region, and the abdomen is retracted and tender. Occasionally an intense nephritis is added. The urine becomes scanty and contains blood and a large quantity of albumin. Death may ensue with all the symptoms of uræmia. There is often slight pyrexia at the height of the attack, but the temperature may be normal throughout the illness. In fatal cases the spleen is found to be swollen, and its enlargement has been detected by percussion during life. Endocarditis and pleurisy have been noted in one case, but their occurrence may be attributed to antecedent rheumatism. As a rule, the articular pains, the petechiæ, and the gastro-intestinal symptoms appear in the sequence given.

Here, as in arthritic purpura, there is a strong tendency to recurrence. After a day or two of the gastro-intestinal symptoms rapid improvement sets in, only to be followed in a few days or weeks by a repetition of the attack. Sometimes a series of such attacks will prolong the illness into several months. Rarely an interval of many months elapses between attacks. Ultimate recovery may be expected as a rule, but it must be remembered that the nephritis may prove fatal, and that in one recorded case death occurred from perforation of the stomach on the fourth day of a second attack.

(5) *Febrile Purpura.*—This form is occasionally seen in young children; it is most common in adolescents, and it is rare after the age of thirty. Males seem to be more affected than females. As with the other forms of purpura, no constant factor can be detected in the history of the patient or of his family.

As a rule, the sudden appearance of petechiæ, or of epistaxis, or of oozing from the gums, brings the patient under observation. On inquiring it is sometimes found that for a few weeks, or even months, the patient has not been in good health; pallor, loss of appetite, and listlessness being noticeable. Whether such a history is given or not, it will generally be seen that the patient is already extremely pale. In rare instances the patient has been previously subject to epistaxis, and he may even have noticed a petechial eruption on a small scale on one or two occasions in the previous year. Sometimes, however, the onset is as sudden as in any specific fever. For example, a telegraph boy, whose history and appearance testified to perfect health, was seized with vomiting and malaise at midday. At 4 P.M. he was obliged to take to his bed. On the next day, without any fresh symptoms, he was unfit to get up. On the following day petechiæ appeared, and death took place in twelve days from the onset.

Whether the onset is sudden or gradual, after the appearance of the first hæmorrhagic symptom the illness proceeds rapidly and uniformly to a fatal end. The petechiæ appear in crops all over the body, thickly or thinly, showing no characteristic distribution. Ecchymoses are common. Of frequent occurrence are tender, bluish-red raised patches, an inch or more in diameter, with a zone of bruising around. On such a patch a bulla containing clear or blood-stained serum is often developed, and this on breaking gives rise to an ulcer from which a thin sanguineous fluid exudes. Such an ulcer may grow deeper by progressive necrosis in spite of all bactericidal treatment.

The mouth is always affected in some degree. Petechiæ can always be seen, and they are here apt to give rise to small grey-capped ulcers. Large sloughy patches are generally found inside the cheeks, and gangrene of the gums at one or more spots is a common occurrence. Neither of these phenomena can be attributed to previous hæmorrhage. Still less can such an explanation be offered as regards a progressive uncontrollable sloughing of one or both tonsils which is not uncommonly observed. Gangrenous ulceration has been known to affect soft palate, epiglottis, and arytenoid folds.

The breath is generally offensive, and it may be exceedingly fœtid. The submaxillary glands are usually enlarged.

Slight continual oozing from the gums is a nearly constant symptom, and the mouth is unclean from the presence of decomposing clot. The gums are pale and not swollen. Epistaxis is common, and it may necessitate plugging of the nostrils. Retinal hæmorrhages are often observed. If sickness occurs, as is often the case, the vomit may contain a little altered blood, but hæmorrhage from the gastro-intestinal and urinary tracts is often entirely absent, and it is never a prominent symptom. Slight albuminuria may appear, probably attributable to the anæmia, and systolic murmurs are often developed at the base and apex of the heart from the same cause. Enlargement of the spleen has been detected by percussion. Joint-pains may be complained of without external evidence of inflammation. The bowels are apt to be loose.

Pyrexia is always a striking feature. It is often present when the patient first comes under observation. Sometimes, however, the first crop of petechiæ is not attended by a rise of temperature, and in such cases a diagnosis of purpura simplex may be made, and a favourable prognosis may be too hastily given. The temperature ranges through 2 or 3 degrees daily, the maximum being generally at least 103°, sometimes 104° or higher. The patient sometimes falls into the typhoid state. Often, however, he does not present the usual febrile aspect, but he is extremely pale and blanched, while the tongue does not grow dry, and is often surprisingly clean. He is generally sleepy, but when roused the mind is clear. He is often singularly hopeful, and even when death is near may complain of nothing but faintness and weakness. The blood presents no change, beyond great diminution of red cells. Leucocytosis is slight or absent. The red cells are usually at least 50 per cent below the normal, and sometimes much below this point. A drop to 390,000 per c.mm. has been observed. It must be noted that the anæmia is often quite out of proportion to the loss of blood, and that it is sometimes extreme when a scanty crop of petechiæ and a little oozing from the gums represent the total amount of hæmorrhage. The amount of hæmoglobin falls equally with the number of red cells.

The real duration of the illness can hardly be estimated, but death commonly occurs during the first, second, or third week after the patient comes under treatment. It is doubtful if recovery ever takes place. The immediate cause of death is generally cardiac failure.

Morbid Anatomy.—After death petechiæ are found to be as widely distributed inside the body as on the skin. In many cases all organs and tissues are thickly strewn with them. Sometimes one part is more affected than another, but the serous membranes and the heart seldom if ever escape. The whole surface of the stomach, and large tracts of the intestine, may be reddened with innumerable petechiæ, which sometimes fuse into extensive hæmorrhagic patches, especially in the rectum. The mesenteric glands are generally pink, and slightly swollen. Hæmorrhage may occur into the brain tissue, and extravasations are often abundant in the pia-arachnoid. Œdema, or broncho-pneumonia, and collapse may be present in the lungs. All viscera, as a rule, are exceedingly pale, though the kidneys after death from Henoch's purpura may present the usual appearance of an acute nephritis. The heart-muscle and liver are soft and pale, and iron-containing pigment may sometimes be demonstrated in the outer zone of the hepatic lobules. After death from febrile purpura the spleen is generally found to be enlarged. In one case it weighed twenty-three ounces. The marrow in the heads of the long bones has occasionally been noted as puriform in appearance, consisting almost entirely of fat, but it is uncertain whether this change is of common occurrence.

Microscopical examination shows that the hæmorrhage occurs mainly by rupture of vessels, and not by diapedesis, though the latter process may play some part. It has been mentioned that the site of the hæmorrhage is generally the lower part of the cutis. In horizontal sections through petechial spots the remains of ruptured vessels may be seen among the extravasated corpuscles in the form of streaks of connective tissue and endothelial plates, which are bent to a common centre.

Treatment. — In symptomatic purpura no special line of treatment can be laid down. If it occurs in association with specific fevers it usually indicates a high degree of toxæmia, and so far as possible this blood-state and its effect on the heart must be met. In the other cases it is sufficient to recognise the cause

In simple purpura little is required beyond rest in bed and tonics, among which arsenic is to be preferred. Great care must be taken from the outset, in view of the fact that the early stage of the graver forms of the disease may be indistinguishable from a simple purpura. In arthritic purpura anti-rheumatic treatment may be adopted, though it is doubtful if the course of the disease is thereby shortened. Complete and prolonged rest in bed is essential. In Henoch's form opium will be found necessary. When danger arises from sheer loss of blood, as in Werlhof's disease, the indication for treatment is plain, but the result is disappointing. Calcium chloride, ergot or ergotin, sulphuric acid, acetate of lead, or turpentine may be tried, with but little result. In the febrile form little can be done beyond combating the cardiac weakness by means of stimu-

lants, strychnia, and inhalation of oxygen, while attempts must be made to check any necrotic tendency in the mouth. Neither large doses of quinine nor injection of anti-streptococcic serum have any influence upon the temperature, and all drugs such as calcium chloride and arsenic are apparently valueless.

Purpuræmia. *See* Blackwater Fever (*Nomenclature*).

Purpurin. — A pigment, also called uroerythrin, which gives the pink colour to urates; also a dye ($C_{14}H_8O_5$) contained in madder (*Rubia tinctorum*).

Purring Thrill. — A trembling sensation felt over the præcordia in some cases of valvular heart disease.

Pus. *See* Suppuration; Appendix Vermiformis (*Appendicitis, Abscess*); Bone, Diseases of (*Osteomyelitis, Local Symptoms, Suppuration*); Fluids, Examination of Pathological (*Pus*); Liver, Tropical Affections of (*Tropical Abscess*); Nose, Accessory Sinuses, Inflammation of (*Latent Empyema, Presence of Pus*).

Pustulant.—A powerful irritant, such as croton oil, which, when applied to the skin, causes acute inflammation accompanied by the formation of vesicles and pustules.

Pustule, Malignant. *See* Anthrax; Malignant Pustule.

Pustules. *See* Eczema; Prurigo and Pruritus.

Pustuloderma.—A skin disease characterised by the formation of pustules.

Putamen.—The outer or basal zone of the lenticular nucleus of the brain.

Putrefaction. *See* Death, Signs of (*Putrefaction*); Labour, Retention of Placenta (*Putrefaction of Retained Fragments*); Medicine, Forensic (*Signs of Death, Putrefaction*); Micro-Organisms (*Putrefaction*); Pregnancy, Intra-Uterine Diseases and Death of the Fœtus (*Putrefaction*); Puerperium, Pathology (*Puerperal Infection, Prophylaxis and Treatment*).

Putrescin. — A poisonous ptomaine, ($C_4H_{12}N_2$) produced in putrid human remains. *See* Immunity (*Immunisation with Toxic Filtrates of Bacterial Cultures*); Ptomaines; Snake-Bites and Poisonous Fishes (*Putrefaction of Fish*); Urine, Pathological Changes in (*Abnormal Nitrogenous Constituents, Diaminuria*).

Putrid Fever. *See* Typhus Fever.

Putrid Sore Mouth. *See* Stomatitis (*Ulcerative*).

Putty, Horsley's. *See* Brain, Surgery of (*Trephining, Operative Details*); Horsley's Putty.

Pyæmia. *See also* Actinomycosis (*Synonyms*); Bed-Sores (*Diagnosis*); Breath (*Odour*); Bronchiectasis (*Cause of Death*); Chorea (*Prognosis*); Colon, Diseases of (*Prognosis*); Hip-Joint, Diseases of (*Pyogenic Diseases*); Kidney, Surgical Affections (*Perinephritic Abscess, Suppurative Pyelonephritis*); Knee-Joint, Diseases of (*Pyogenic Diseases*); Larynx, Affections of Cartilages (*Perichondritis*); Leucocytosis (*Infective Conditions*); Liver, Tropical Diseases (*Abscess*); Malaria (*Diagnosis*); Meningitis, Epidemic Cerebro-Spinal (*Diagnosis*); Micro-Organisms; Nose, Acute Inflammation (*Suppurative Rhinitis*); Puerperium, Pathology (*Infection, Pyæmia*); Purpura; Retina (*Hæmorrhages in Pyæmia*); Septicæmia; Stomach, Diseases of (*Infective Gastritis*); Suppuration; Teeth (*Alveolar Abscess*); Typhoid Fever (*Diagnosis*); Urethra, Diseases of (*Gonorrhœa*).— The terms sapræmia, septicæmia, and pyæmia have been applied to various degrees of severity of blood-poisoning. By sapræmia is meant the *presence in the blood of a soluble toxic substance* absorbed from a focus of disease on one of the free surfaces of the body, this disease being induced by pathogenic bacteria. Septicæmia means the presence in the blood not only of these toxic substances, but *the presence in that fluid of the bacteria themselves*; only rarely can the presence of these bacteria in the blood be satisfactorily demonstrated. In pyæmia we have a further grade of severity, in *the tendency to secondary infections of different organs and tissues*—the bacteria passing out from the circulation into the tissues, forming local abscesses at the site of deposit. The pathological appearances induced by these various degrees of infection do not call for description here, other than to say, that every grade of severity is met with, from the simple cloudy swelling and analogous changes in the special tissues to severe suppuration in the organs, serous membranes, and other tissues. Clinically these infections may be acute or chronic. As a rule, the more severe types of septicæmia and pyæmia are associated with some definite breach of surface on the skin or one of the mucous surfaces of the body. As illustrative examples we may quote septicæmia or pyæmia resulting from the occurrence of sepsis at a surgical operation, or during the management of labour. The causes of this systemic infection will be found described in various articles throughout this work; see especially "Puerperium," "Micro-Organisms," "Bones, Diseases of." It must suffice to say here that in many cases it is found that these infections are frequently found to be due to the pathogenic action of such bacteria as bacillus coli communis, staphylococcus pyogenes aureus, and streptococcus

pyogenes—bacteria which under normal circumstances exist in the body as saprophytes. The practical question which presents itself is therefore: What are the agencies by which the tissues, *e.g.* of the alimentary tract, skin surface, respiratory tract, etc., are protected from the injurious action of these bacteria? This question is a most important one, and receives much less attention than it merits. Its undoubted complexity should not interfere with our giving it thoughtful attention. The problems of general and of local immunity are probably the most important questions in present-day medicine and surgery; these have been fully discussed in the general article on "Immunity" in an earlier volume. The future will probably show that the bone-marrow function calls for a much closer and more minute investigation in regard to the problems of subacute and chronic septicæmia and pyæmic condition (see also "Rheumatism").

Pyarthrosis.—The presence of pus in a joint. *See* Joints, Diseases of (*Pyogenic*).

Pycno-.—In compound words *pycno-* or *pykno-* (Gr. πυκνός, firm or thick) means thick or condensed; *e.g. pycnophrasia* (thickness of speech) and *pycnosis* (a condensation or inspissation of an organ or a fluid).

Pyelitis.—Inflammation affecting the pelvis and calyces of the kidney (Gr. πύελος, a vessel or trough). *See* Bladder, Injuries and Diseases of (*Chronic Cystitis, Diagnosis*); Kidney, Surgical Affections of (*Floating Kidney, Kidney Injuries, Pyelitis, Stone*); Liver, Tropical Abscess (*Diagnosis*); Malaria (*Diagnosis*); Typhoid Fever (*Complications, Pyelitis*).

Pyelo-.—In compound words *pyelo-* (Gr. πύελος, a vessel) means relating to the pelvis of the kidney; *e.g. pyelocystitis* (inflammation of the bladder and of the pelvis of the kidney) and *pyelolithotomy* (removal of a calculus by incision into the pelvis of the kidney).

Pyelonephritis. *See* Kidney, Surgical Affections of (*Suppurative Pyelonephritis*).

Pyelotomy.—Incision into the pelvis of the kidney. *See* Kidney, Surgical Affections of (*Operative Procedures*).

Pygo-.—In compound words *pygo-* (Gr. πυγή, the buttocks or rump) means relating to the gluteal region or breech, *e.g. pygomelus* (a teratological type of double monster in which one or two rudimentary limbs are attached to the gluteal region of the autosite).

Pygopagus.—A double monster consisting of two individuals united by the sacrum and coccyx. *See* Teratology (*Double Monsters*).

Pykno-. *See* Pycno-.

Pyle-.—In compound words *pyle-* (Gr. πύλη, a gate or port) means relating to the portal vein, *e.g. pylephlebectasis* (dilatation of the portal vein).

Pylephlebitis.—Inflammation of the portal vein. *See* Appendix Vermiformis (*Appendicitis, Results*); Gall-Bladder and Bile Ducts, Diseases of (*Cholangitis*); Liver, Tropical Abscess of (*Diagnosis*); Liver, Diseases of (*Pylephlebitis, Portal Thrombosis*).

Pylethrombosis.—Thrombosis of the vena portæ. *See* Liver, Diseases of (*Pylephlebitis, Portal Thrombosis*).

Pylogosin, Leber's. *See* Suppuration (*Bacterial Products*).

Pylor-.—In compound words *pylor-* (Gr. πυλωρος, a gate-keeper) means relating to the pylorus.

Pyloralgia.—Pain in the pyloric end of the stomach.

Pylorectomy.—Excision of the pylorus. *See* Stomach, Surgical Affections of (*Operations on the Stomach, Resection of the Pyloric Segment*); Stomach and Duodenum, Diseases of (*Treatment of Dilatation of the Stomach, Surgical*).

Pyloric.—Relating to the pylorus; *e.g.* the *pyloric orifice, artery, plexus,* and *vein. See* Stomach and Duodenum, Diseases of (*Anatomy*).

Pylorocleisis.—Obstruction of the pylorus.

Pylorodiosis.—Mechanical stretching and dilatation of the pylorus. *See* Stomach, Surgical Affections of (*Operations*).

Pyloroplasty.—Plastic surgery as applied to the pylorus. *See* Stomach, Surgical Affections of (*Operations*).

Pyloroptosis.—Downward displacement of the pyloric end of the stomach. *See* Indigestion (*Symptoms*).

Pylorospasm.—Spasmodic contraction of the pyloric end of the stomach.

Pylorostenosis.—Constriction or stenosis of the pylorus.

Pylorus. *See* Gall-Bladder and Bile Ducts, Diseases of (*Tumours of the Gall-Bladder, Diagnosis*); Gastro-Intestinal Disorders of Infancy (*Congenital Hypertrophy of the Pylorus*); Liver, Diseases of (*Acquired Deformities, Constriction Lobe, Diagnosis*); Stomach and Duodenum, Diseases of (*Anatomy, Morbid Anatomy, General and Special Symptomatology*); Stomach, Surgical Affections of (*Pyloric Obstruction, Cancer,* etc.).

Pyo-.—In compound words *pyo-* (Gr. πυός, pus, or the first milk) means relating to pus; *e.g. pyocenosis* (discharge of pus), *pyocyte* (a pus corpuscle), *pyopericardium* (pus in the pericardium), *pyopoiesis* (pyogenesis), *pyorrhœa* (a purulent discharge), *pyoscheocele* (a suppurating scrotal hernia), etc.

Pyocyanin.—A blue colouring matter ($C_{14}H_{14}NO_2$) derived from blue-coloured pus (bacillus pyocyaneus), becoming yellow on oxidation (*pyoxanthose*). *See* Fluids, Pathological Examination of (*Pus*); Pigments of the Body and Excreta (*Bacterial Pigments, Pyocyanin*).

Pyodermitis.—A skin disease characterised by the production of pus. *See* Skin, Bacteriology of (*Pyodermia*).

Pyogenesis. — The formation of pus; pus-forming bacteria are termed *pyogenic*. *See* Bladder, Injuries and Diseases of (*Inflammation, Causes*); Knee-Joint, Diseases of (*Tuberculous Disease, Formation of Abscesses*).

Pyohæmothorax. — The accumulation of pus and blood in the pleural cavity.

Pyoktanin.—Methyl violet and methylene blue have both been called pyoktanin (from their antiseptic properties), and have been used for antiseptic purposes in cystitis, diphtheria, conjunctivitis, etc.

Pyometra.—The presence of pus in the uterine cavity. *See* Uterus, Malignant Tumours of (*Cancer of Cervix, Results, Retention of Secretion*).

Pyonephrosis.—The presence of pus in the pelvis (and substance) of the kidney. *See* Ascites (*Distinction from other Conditions*); Kidney, Surgical Affections of (*Movable Kidney, Perinephritis, Pyonephrosis*); Uterus, Non-malignant Tumours of (*Fibroids, Complications*).

Pyophthalmus.—A collection of pus within the eyeball.

Pyopneumothorax.—The presence of pus and air in the pleural cavity. *See* Diaphragm, Surgical Affections of (*Inflammatory, Subphrenic Abscess, Diagnosis*); Pleura, Diseases of (*Pneumothorax*).

Pyorrhœa.—A purulent discharge. *See* Hæmatemesis (*Causes*); Mouth, Injuries and Diseases of Jaw (*Chronic Suppuration, Pyorrhœa Alveolaris*); Teeth (*Pyorrhœa Alveolaris*).

Pyosalpinx.—The presence of pus in the Fallopian tube (*salpinx*). *See* Fallopian Tubes (*Purulent Salpingitis*); Gonorrhœal Infection (*Complications*); Menstruation and its Disorders (*Menorrhagia and Dysmenorrhœa, Causes*); Pelvis, Hæmatocele and Hæmatoma (*Diagnosis*).

Pyoscope.—An instrument for estimating the richness of milk in cream (Gr. πυός, the first milk, or colostrum).

Pyothorax.—Pus in the pleural cavity; empyema. *See* Pleura, Surgical Affections of (*Empyema*).

Pyoxanthose or Pyoxanthin.—A yellow pigment found in pus, derived from *pyocyanin* (*q.v.*).

Pyramid.—The term applied, in Anatomy, to various structures in the body (belonging to the kidney, nervous system, etc.) which have the pyramidal form; *e.g.* the *pyramids of Malpighi* and *of Ferrein*, and the *pyramids of the medulla*.

Pyramidalis. — The name given, in Anatomy, to certain muscles having a pyramidal form, such as the *pyramidalis abdominis* and the *pyramidalis nasi*.

Pyrenæmia.—The state of the blood in which the red corpuscles have nuclei (Gr. πυρήν, a kernel or nucleus).

Pyrenin.—A modification of the karyoplasm (modified protoplasm) of the nucleus of the cell.

Pyrethri Radix.—Pellitory root or *pyrethrum*, the dried root of *Anacyclus pyrethrum*, causes in the mouth a burning or pricking sensation followed by the secretion of saliva; it consists of inulin and volatile oils and resins; there is an official preparation, the *tinctura pyrethri*; and the drug is sometimes added to tooth-powders.

Pyretic.—Febrile (Gr. πυρετός, burning heat); the term is more familiar in the word *antipyretic*.

Pyreto-.—In compound words *pyreto-* (Gr. πυρετός, burning heat) means febrile or relating to fever; *e.g. pyretogenesis* (the production of fever), *pyretology* (the science of fevers).

Pyrexia.—Fever, or the febrile state (Gr. πυρέσσω, I am feverish). *See* Abortion (*Causes*); Alcohol (*Indications*); Anæmia, Pernicious (*Symptoms, Temperature*); Children, Clinical Examination of (*Temperature, Pyrexia*); Heart, Affections of Myocardium and Endocardium (*Etiology, Thermal*); Invalid Feeding (*Food in Pyrexial States*); Joints, Diseases of (*Tuberculous Disease, Pyrexia*); Lung, Tuberculosis of (*Constitutional Symptoms*); Pediculosis (*Pediculosis Corporis, Symptoms*); Pulse (*Characters in Pyrexia*); Rheumatism in Children (*Nervous Affections, Hyperpyrexia*); Suppuration (*Symptoms, Constitutional, Pyrexia*); Typhoid

Fever (*Symptoms*); Undulant Fever (*Symptoms, Pyrexia*).

Pyridine. *See* Alkaloids (*Pyridin*).

Pyriform or Pyriformis. — Pear-shaped; *e.g.* the pyriform lobe (lobus pyriformis) of the brain, the pyriformis muscle and fascia, etc. *See* Physiology, Neuro-muscular Mechanism (*Cerebrum*).

Pyrmont. *See* Balneology (*Germany, Chalybeate Waters*); Mineral Waters (*Chalybeate*).

Pyro-.—In compound words *pyro-* (Gr. πῦρ, fire) is a prefix signifying relating to heat or produced by the agency of heat; *e.g. pyroacetic ether* (acetone formed by the dry distillation of acetates), *pyrogenesis* (the generation of fever), *pyroligneous acid* (pyroacetic acid obtained by the dry distillation of wood), *pyroarsenates*, etc.

Pyrocatechin. — Ortho-dihydroxybenzene ($C_6H_6O_2$), a substance obtained by the dry distillation of catechu; it has been used as an antipyretic (dose, 1 to 2 grains), and it is sometimes found in the urine. *See* Urine, Pathological Changes in (*Aromatic Substances, Ethereal Sulphates*).

Pyrodin.—A coal-tar derivative consisting chiefly of hydracetin or acetylphenylhydrazine ($C_6H_5.C_2H_3O.N_2H_2$); it has been used as an antipyretic (dose, ½ to 3 grains) and in psoriasis.

Pyrogallic Acid.—Acidum pyrogallicum or trioxybenzol or pyrogallol ($C_6H_6O_3$) is a non-official preparation, obtained from gallic or tannic acid by heat; it is used, in the form of an ointment (*Jarisch's*), in cases of chronic psoriasis and in ringworm. *See* Jarisch's Ointment; Psoriasis; etc.

Pyrogallol. *See* Pyrogallic Acid.

Pyromania.—Incendiarism due to insanity. *See* Insanity, Nature and Symptoms (*Impulsiveness*).

Pyrophobia.—An insane dread of fire. *See* Insanity, Nature and Symptoms (*Impulsiveness*).

Pyrosal.—Salicylacetate of antipyrin, an antipyretic drug, recommended in fever and neuralgia (dose, 8 grains).

Pyrosis.—Waterbrash or heartburn. *See* Stomach and Duodenum, Diseases of (*General Symptomatology, Eructations*).

Pyrosoma.—One of the hæmosporidia; the *pyrosoma bigeminum* is the cause of Texas fever. *See* Malaria (*History*); Parasites (*Protozoa, Hæmosporidia*).

Pyrotoxin. *See* Temperature (*Causes of Fever*).

Pyroxylin. — Dinitrocellulose ($(C_6H_8(NO_2)_2O_5)$), is cotton-wool which has been soaked in a mixture of sulphuric and nitric acids and dried. *See* Collodion; Gossypium; etc.

Pyrrhol. *See* Physiology, Protoplasm (*Products of Decomposition, Pyrrhol Derivatives*).

Pyuria.

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See also Children, Clinical Examination of (*Urine, Pyuria*); Kidney, Surgical Affections of (*Stone in the Kidney, Symptoms*); Liver, Tropical Abscess of (*Prognosis from Pus in the Urine*).

The appearance of pus in the urine, like the presence of blood, is a symptom of many different lesions in the urinary tract. It may emanate from any portion of the apparatus, or may gain an entrance to it from neighbouring parts. Pus appears in amounts to be appreciated by the microscope only, or it may escape in very large quantities, so as to form a considerable bulk of the evacuation. It is necessary to remark here upon the methods used in the detection of pus, and the means employed to distinguish it from other urinary deposits.

The *gross physical characters of pus* vary considerably. It may be present in an acid urine in small quantity, when it deposits as a slight cloud at the bottom of the test glass; it may be mixed with mucus, and elude detection except by the microscope; when abundant and in an alkaline urine it may form a copious yellowish white creamy sediment, or it may be curdy and contain flakes. The appearance of pus is only a sign. It is not the physical properties of the pus that is of importance, but rather the bacteriological conditions which are associated with its presence, and unfortunately the former afford no clue to the latter. Besides detecting the presence of pus in the urine the clinical observer must determine the micro-organisms giving rise to the formation of the discharge. By the microscope alone the presence of bacteria may be demonstrated, but in order to reveal their identity cultivations are necessary, and perhaps these methods may require to be supplemented by inoculation experiments upon animals. The detection of pus in the urine is seldom of itself sufficient to guide the surgeon to a conclusive diagnosis, although by it alone information may be obtained to assist him considerably in his inquiry. But when the special characters of the pus are studied in conjunction with the circumstance attending its escape, and in view of the general features of the case, little difficulty will be experienced in discovering the nature and position of the morbid lesion.

Examination of the Urine.—The appearance imparted by the addition of pus varies according to the quantity present, and the character of the other morbid constituents in the urine.

In cases where the pyuria is due to septic lesions of the kidney only, the pus appears as a thick, opaque, creamy sediment, which on standing is rapidly and almost completely precipitated from an acid urine; the supernatant fluid is comparatively clear, is free from mucus, and does not decompose rapidly. On the other hand, where the pus is derived from the bladder alone, or in conjunction with a renal lesion, pus is not deposited quickly; when it falls it is precipitated along with glairy mucus, the urine in which it remains partly suspended is alkaline, and decomposition often begins before the urine escapes from the bladder. The time at which the pus appears in the stream should be ascertained if possible. The appearance of pus at the beginning of micturition, during the whole flow, or at the end of the act only, lends additional significance to the symptom. The precaution to ascertain the moment at which morbid constituents appear in the stream has already been referred to in connection with hæmaturia. Sir Henry Thompson, in his *Clinical Lectures on Diseases of the Urinary Organs*, 8th edition, pages 12 and 452, gives the following valuable suggestions:—"Whenever you want a specimen from your patient to examine, do not tell him to send you a bottle of it passed in the usual way, or you will get a mixture often of doubtful value. What you require is the secretion of the kidneys, plus only anything there may be in the bladder; but you also have to avoid the presence in it of any secretion which originated in the urethra. Make a point of demanding that the patient should first pass two or three tablespoonfuls through the urethra, so as to sweep out whatever may happen to be there (which may be thrown away, or be put into a separate bottle), after which you will get a pure specimen for examination—at any rate one of which you will know the source. You will have the renal secretion, plus only whatever deposit may be produced in the bladder. Suppose the patient has gleet or chronic prostatitis, there will then be a quantity of muco-purulent matter in the urethra. If all this be carried into one vessel with the urine, how will you determine the different products, and decide, by the eye or by the microscope, what has come from the urethra, what from the prostate, and what from the kidneys? You cannot do it; but if you get rid of the source of error by flushing the urethra, so to speak, that is, by passing the first two or three tablespoonfuls into a wine-glass, while all that follows is passed into a separate vessel, such as a tumbler, you will have in the latter a sample of urine that can be relied upon for examination.

"The necessity for avoiding admixture of urethral products with urine, in order to ensure a pure specimen, has been enjoined, and the mode of attaining the object by the use of two glasses. It is sometimes quite as essential to avoid its admixture with products of the bladder. And it is not possible to make an absolute diagnosis in some few exceptional cases without following the method in question. When, therefore, it is essential to obtain a pure specimen of the renal secretion free from the presence of vesical products, I pass a soft flexible catheter, of medium size, into the bladder, the patient standing, draw off all the urine, carefully wash out the viscus by repeated small injections of warm water, and then permit the urine to pass, as it will do, *guttatim*, into a test-tube, or other small glass vessel, for purposes of examination. The bladder ceases for a time to be a reservoir; it does not expand, but is contracted round the catheter, and the urine percolates from the ureters direct. The ureters are virtually lengthened as far as to the glass. Thus is obtained a specimen which, for appreciating albumin, for determining reaction, and for freedom from vesical pus and even blood, and from cell-growths of vesical origin, is of considerable value, and has sometimes furnished me with the only data previously wanting to accomplish an exact diagnosis."

The clinical significance of pyuria in many respects resembles that of hæmaturia, but differs from it in so far that an accurate diagnosis of the portion of the urinary tract from which the pus comes is more difficult to make.

Diagnosis of Origin

Pus from the urethra, as in hæmaturia from the same situation, escapes independently of micturition, or is only mixed with the first few drops of urine; in fact, together with the presence of pain in the region, is usually sufficient to determine its origin. In such instances the quantity of pus is usually small and appears in flakes or threads. These are casts of ducts leading into the urethra, or threads of pus and macerated epithelium from granular patches swept away in the first portion of the stream. These urethral casts or shreds must not be mistaken for somewhat similar discharge occasionally observed in tuberculosis of the urinary tract. When urethral in origin they can be expressed from the canal before and after micturition. When the inflammation is situated in the posterior part of the urethra it may give rise to vesical irritation and some difficulty to micturition. The diagnosis can generally be cleared up by pressure upon the urethra in the perineum or rectum towards the end of micturition when pus is found to exude.

It is otherwise in *catarrh of the bladder*, in which case the pus only escapes mixed with the urine, is more or less abundant, and is accom-

panied by a number of large vesical epithelial cells. Vesical catarrh is generally associated with considerable pain on micturition and inability to retain urine for any length of time. A small quantity of pus in an acid urine is not necessarily of much significance, as it may be due to the slight catarrh seen so frequently in uterine and ovarian disease, but it may also be one of the earlier symptoms of tuberculosis. When the irritation is more severe, and especially when due to the action of micro-organisms, not only is the quantity of pus increased, but much mucus may be secreted. So long, however, as decomposition does not occur, the pus and mucus separate as distinct deposits on standing, but if the urine becomes alkaline before evacuation the pus becomes thick and glairy, and remains suspended along with the mucus. To establish the fact that there is cystitis is not sufficient; the cause of the altered condition must be determined, and this may be done by a cystoscopic examination.

If it has been ascertained that there is no primary urethral lesion and no disease of the bladder, neither inflammation nor suppuration of the prostate, no pelvic or other abscesses opening into the urinary tract, then we must proceed to inquire whether the pus is proceeding from one or from both ureters. This may be done by inspecting the bladder with the cystoscope or by catheterising the ureters.

Having discovered that pus is flowing with the urine from the ureters, the next point to clear up is the nature of the renal lesion. The most important diseases of the kidney which are characterised by pyuria are pyelitis, pyonephrosis, with or without calculus, suppurative nephritis, tubercular disease, and cystic disease when the cyst has suppurated.

The question may now be asked: Is there, by observing the urine, any means of distinguishing one form of renal pyuria from another? To some extent the microscopical appearance of the corpuscles may serve as a guide. When the pus-cells are round, well-developed, the contents of the corpuscles clear, and on addition of acetic acid show well-marked nuclei, the presumption is that they are of recent origin and have formed on a mucous surface. But if the cells contain a considerable quantity of finely granular particles, which impart a cloudy appearance to the protoplasm, if the cell-wall be irregular and ill-defined, and the nuclei few in number and small in size, the suppuration is probably deep seated and due to chronic inflammation.

When pyuria is a consequence of *inflammation* of the *pelvis* and *calices of the kidney*, being mixed with an acid urine, the pus on standing usually precipitates rapidly, separates clearly from the supernatant fluid, and if the inflammation be very acute, or if a calculus be present, the pus may occasionally be mixed with blood. A careful record must be kept not only of the relative, but also of the absolute quantity of pus, as sudden and marked variation in the amount is one of the most certain indications of partial or complete obstruction of the ureter from which the pus is flowing. If the disappearance of pus from the urine be associated with increase in the lumbar swelling, then followed by the sudden escape of a large quantity of purulent urine coincident with a subsidence of the swelling, then it is clear that the ureter has been blocked for a time and its patency restored.

As the result of *calculous pyelitis*, prior to the appearance of pus, the urine is usually observed to contain an abundant quantity of mucus, which forms a bulky, tenacious mass. So long as the urine remains acid the mucus is readily deposited from the urine. This is especially the case when the calculus is composed of oxalate of lime. Before pyuria is observed microscopic examination of the deposit may demonstrate the presence of large spheroidal granular cells, degenerated epithelium, and a few blood corpuscles. When the pyelitis, which in the early stage is simply the result of mechanical irritation, has existed for some time, the quantity of pus increases and the mucus disappears. So long as the urine remains acid and no vesical disturbance arises, micturition is not unduly frequent, and the urine has not an offensive odour. But if there be frequent or considerable accumulations in the pelvis of the kidney as a result of obstruction, the fluid so retained is liable to septic infection; and if the organisms have the property of decomposing urea, the urine rapidly becomes alkaline and glairy, has an offensive odour, and is in almost all respects similar to the urine in cystitis.

When *suppurative nephritis* arises as a complication of the retention of decomposing urine, or as a sequence to a chronic pyelitis, the course of the disease is usually very rapid, and the diagnosis is not difficult. The history of some obstruction to the flow of urine is usually found, whether it be from stricture of the urethra, enlarged prostate, atony of the bladder, vesical calculus, tumours of the bladder, tuberculosis, disease of the spine, or other cause by which the bladder is kept in a distended state and its contents rendered liable to decomposition.

When decomposition of urine in the pelvis of the kidney occurs, pyelonephritis soon manifests itself, sometimes, but not usually, by severe pain in the loins, aggravated by pressure upon the abdominal walls over the kidney. Although pain in the renal region is rare, the patient suffers from spasm at the neck of the bladder and pain along the tract of the ureter.

The urine does not present any characters of diagnostic value as regards the situation of the lesion, and varies much according to the individual case. When the inflammation primarily

involves the pelvis, and has spread rapidly from it to the kidney tissue, the urine, although purulent, remains acid for some time, and is less abundant than normal. The urine may, however, present the characters indicative of chronic Bright's disease, namely, polyuria and albuminuria, with diminution in the quantity of urea and the presence of tube casts.

Pyelonephritis is usually the result of an unhealthy state of the bladder. Consequently in that disease the urine, although it may be acid when it escapes from the ureter, becoming mixed with the contents of the bladder decomposes, is passed alkaline, has an offensive odour, and contains a large quantity of mucus, epithelium, pus, and triple phosphates. When there are no acute or chronic organic changes in the kidney, the quantity of albumin is regulated by the amount of pus or blood; but, as not infrequently chronic Bright's disease also exists, there is an albuminuria independent of these constituents, so that no proportionate relationship exists between the amount of pus or blood and the quantity of albumin.

The diagnosis of pyelonephritis from suppurative nephritis must rest largely upon a careful consideration of the etiological facts of the case. The constitutional symptoms of the two diseases are practically the same. In the former, however, pyuria precedes, in the latter it succeeds the other symptoms. If the abscess in the kidney ruptures externally, or forms a perinephritic abscess throughout the course of the case, pus may not appear in the urine; but should the abscess attain a considerable size, and rupture into the pelvis, ureter, or bladder, the sudden appearance of a large quantity of pus in the urine may clear up the diagnosis.

In *pyonephrosis* there is evidence both of suppuration and of obstruction. In some cases suppuration precedes the obstruction; in others, the opposite sequence of events occurs, as when a hydronephrosis becomes inflamed, while in a third class the two conditions develop *pari passu*.

In pyonephrosis there is a characteristic renal swelling, elastic, fluctuant, and varying in size from time to time, much in the same way as in hydronephrosis. Together with these symptoms there is a discharge of pus in the urine, or evidence of its retention, manifested by increase in the renal swelling, and more severe pain in the lumbar region, which is aggravated by pressure in front, but relieved by pressure from behind. There may be also symptoms of a more urgent nature, indicative of systemic poisoning, either from non-elimination of the urinary constituents or from absorption of infective material.

In pyonephrosis the quantity of pus in the urine varies from day to day, according to the posture the patient occupies and to the nature of the obstruction. The changes in the condition of the urine are determined by the same circumstances as those noticed in cases of pyelitis and pyelonephritis.

In *cystic disease* the urine has the characters of chronic interstitial nephritis; it is pale, copious, of low specific gravity, and may contain coarse granular tube casts. There may be copious hæmaturia, and should the cysts suppurate or pyelitis be established, pus may escape through the bladder, and in hydatid cysts of the kidney echinococci or hooklets may appear in the urine.

In *tubercular disease* the urine furnishes important information, and in all cases the urine should be carefully examined prior to the employment of instruments to explore the bladder or ureters. Although tuberculous bacilli are classified as non-pyogenic, and properly so, in many cases of tuberculous disease before mixed infection has occurred large numbers of leucocytes not distinguishable by the microscope from pus may be found. In pure tuberculous disease the urine is acid and free from pyogenic microbes. For diagnostic purposes it is therefore necessary to draw off, through a sterilised catheter, a quantity of urine and to make cultures. The presence of pyogenic micro-organisms does not exclude the possibility of the disease being tuberculous, but their absence greatly strengthens the presumption.

As the disease advances the odour of the urine as a rule becomes more and more offensive; the fluid deposits large quantities of mucus and triple phosphates, and on examination it is found to be highly albuminous. Occasionally, while the urine is pale and of low specific gravity, there may be evidence of retention on the diseased side, as shown by increase in the renal swelling, with perhaps indistinct fluctuation and pain, accompanied by general constitutional disturbance, or there may be complete suppression, death being ushered in by uræmic symptoms.

In addition to the diagnostic points already indicated, the detection of the tubercle bacillus in the urinary debris is of great value. It is when tuberculous lesions begin to break down that tubercle bacilli are most abundant, and sometimes their number is very large. Several examinations are required before it can be concluded from negative results that the case is not tuberculous.

Besides the detection of the tubercle bacillus it is necessary to determine whether the disease is on one or on both sides. Several times the writer has had occasion to examine by ureteral catheterisation cases of tuberculous pyelitis in which it was considered of importance to determine the extent to which the disease had involved one or both kidneys. Catheterisation can only determine the organ to which the tuberculous disease is limited; it cannot indicate the extent of the tuberculous lesion in the

affected organ, but to make sure that one kidney is free from disease is a point of importance. The first time he succeeded in doing this was in 1886, when a case was examined in which it was clearly shown that the tuberculous lesion was limited to one kidney.

Quadrant.—The fourth part of a circle or of any area resembling a circle; *e.g.* the brim of the pelvis, in which the space between two of the points of Capuron is a quadrant.

Quadrate or Quadratus.—Four-sided; *e.g.* the quadrate lobe of the liver, the quadrate bone, the quadratus femoris muscle, the quadratus lumborum muscle, etc.

Quadri-.—In compound words *quadri-* (Lat. *quatuor*, four) means four times; *e.g. quadricornate*, *quadridigitate*, etc.

Quadriceps.—Having four heads; *e.g.* the large extensor muscle of the thigh (*quadriceps extensor cruris*), which includes the rectus femoris, the vastus externus and internus, and the crureus.

Quadrigeminal.—In two pairs, or fourfold; *e.g.* the *corpora quadrigemina of the brain*, the *quadrigeminal pulse*, etc. *See* Pulse (Fig. 74).

Quadriurates. *See* Physiology, Excretion (*Urine, Nitrogenous Substances, Uric Acid*); Uric Acid (*Chemical Characters*).

Quadruplets.—Four at a birth. *See* Pregnancy, Multiple (*Varieties*).

Quain's Degeneration.—Fatty degeneration of the cardiac muscle; Quain's fatty heart.

Quarantine. *See also* Disinfection (*Quarantine*).—The word is derived from the forty days which formerly was the period during which persons were detained when coming from an infected country.

The system was originally introduced with the idea of protecting a country from the danger of importation of disease, a danger which is practically in direct proportion to the extent of its trade. Regulations for this purpose are sanctioned in principle by international law, which, recognising the necessity for the constant interchange of goods and commodities between various states, allows each one to protect itself in any way it may deem fit or expedient. Thus, whatever restrictions any one state may adopt, these must be submitted to by all others, as the only conditions under which trade with such state is possible.

Hence the extreme importance of the subject from a commercial point of view. The history of quarantine in this country is that of a long and fierce struggle between the two great interests of free-trade intercourse and the necessity of guarding against the risk of introducing disease arising from such intercourse.

The earliest mention of any system of defence against infectious disease is found in the marvellous and complete sanitary code promulgated by the ancient Jewish lawgiver Moses under divine guidance.

In early days the period of detention considered necessary for safety was a purely arbitrary one, but as medical science developed an attempt was made to limit its duration to some extent in proportion to the length of the incubation period of the disease against which it was directed.

The essential point of quarantine from its earliest inception up to its modern development was to estimate the danger (and, therefore, the precautions to be taken) according to the state of health of the port from which a vessel had arrived, without any reference to the condition of the vessel and its inhabitants as regards health and sickness.

The earliest definite codes of quarantine directed against plague were issued by the Eastern emperors during the sixth century, while probably the most elaborate system of marine quarantine was established by the Venetians in the twelfth century, when they obtained sanction from the Pope to trade with the infidels of Alexandria and Aleppo. According to this code, all merchants and others coming from the Levant were compelled to remain in the house of St. Lazarus for forty days before entering the city.

In this country the first mention of marine quarantine was in 1636, when the Royal College of Physicians, in "Directions" issued by them, enacted that "care be taken that neither man nor goods may come from any suspected places without a certificate of health, or else either to be sent suddenly away or to be put to the pest-house for forty days according to the custom of Italy."

The first Quarantine Act in England was passed in the ninth year of Queen Anne. Up to that time all quarantine regulations had been issued by Orders in Council without the intervention of Parliament. But the quarantine regulations were never constant, and from their uncertainty and the increasing changes in connection with their enforcement, continually became more and more burdensome to trade.

A report of a select committee on the foreign trade of the country in 1824 mentions that the quarantine charges in the case of a vessel with a clean bill of health amounted to 7s. 6d. per ton, and in the case of a foul bill to 15s. per ton, 1s. extra being paid in London. These charges, moreover, were levied on the gross tonnage of the vessel whether she carried a full cargo or no, one instance being given in which they exceeded 90 per cent of the value of the

cargo, although there had been no sickness on board the vessel.

The last Quarantine Act was that of 1824, which placed the whole system in the hands of the Privy Council, and though subsequently the actual working was carried out by Her Majesty's Customs, it remained in their hands till its final abolition by the Public Health Amendment Act, 1896.

But for a long period it had been in abeyance, remaining only on the statute book in order to avoid interference with our shipping by foreign powers. While nominally the power was in the hands of the Privy Council, who had no machinery for the purpose, the actual work of protection of the country against infection from abroad was under the control of the port sanitary authorities and their medical officers.

Port sanitary authorities were first constituted by the Public Health Act of 1872, and their powers have been slowly but gradually extended. In 1893, at the International Sanitary Congress, when Great Britain was so ably represented by the late Sir Richard Thorne Thorne, the question advanced one great step.

To his able advocacy of medical inspection as a substitute for quarantine, involving as it does the consideration of the health of the crew of a vessel and the sanitary condition of the vessel itself, rather than the health of the port from whence she has come, this conference in a great measure owed its successful results.

All the great European Powers finally agreed to the convention. The main points were, that in the case of "infected" vessels the sick were to be removed to hospital, the healthy landed for observation for a period not exceeding five days, and the vessel disinfected. "Suspected" vessels were to be subject to medical inspection, and disinfection of such articles as considered necessary; drinking water to be changed, and the bilges disinfected. "Healthy" vessels were to be admitted to free "pratique" whatever the nature of their bills of health.

An "infected vessel" was defined as one on which a case had occurred within seven days of arrival, while a "suspected" vessel was one on which cases had occurred on the voyage, but not within seven days.

Sir Richard Thorne Thorne, agreeing to the above on behalf of this country, expressly stipulated that observation should not necessarily imply detention, but that this would be covered by registration and notification of the addresses of all persons on board such vessels.

In 1896 the Public Health Amendment Act finally abolished quarantine, and placed the whole system of medical inspection in the hands of port sanitary authorities under the control of the Local Government Board. A Local Government Board order soon followed, defining the powers of port sanitary authorities and regulating their procedure. This order, though modified in several details, still practically governs the system. It defines an "infected" vessel as one on which cholera, yellow fever, or plague has occurred during the voyage, or during the stay of a vessel in the port of departure, or in a port on the course of such voyage.

The officer of customs who visits the vessel, if he has any reason to suspect the vessel to be so infected, has power to detain her, and must give notice to the Medical Officer of Health, who shall thereupon visit her. There is, however, an important section which gives power to the medical officer to visit any vessel for the purpose of ascertaining if she is infected, whether she has been visited by the customs officer or not. Under this section the Medical Officer of Health can visit every vessel coming from an infected port.

The only satisfactory method of working this system is in conjunction with the customs officer at the appointed boarding station.

In this way all the necessary precautions for the protection of the public health can be taken without hampering commerce by requiring an incoming vessel to be detained for two separate visits. This means, if properly worked, that a Medical Officer of Health must be always on duty, and accompany the customs officers on their visit for fiscal and statistical purposes. In the port of London this is carried out by having a special staff of boarding medical officers who are on duty in turn for twenty-four hours every third day.

Whether on his own initiative or informed by the customs officer, the medical officer visits a a suspected vessel and examines it. If he finds the vessel to be infected, he serves a certificate to that effect on the master, who thereupon must moor his vessel in such place as required, and must carry out the instructions of the medical officer. No person may leave the vessel till he has been medically examined; those found to be suffering from cholera, yellow fever, or plague are removed to hospital under the control of the medical officer, any suspected cases being detained for any period not exceeding two days for the purposes of diagnosis.

All those found healthy are required to furnish their names and addresses at their intended places of destination, and are then allowed to leave. These names and addresses are forwarded to the sanitary authorities of the places of destination concerned, and thus afford each authority an opportunity of keeping itself informed as to the health of such persons for so long a period as may be considered advisable.

By the terms of the order, the vessel and all effects are disinfected by the master of the vessel in accordance with the instructions of the medical officer, though in practice the disinfection is at once carried out by the officers of the sanitary authority.

There are also special powers to deal with

immigrants and persons of this class who are in a filthy or otherwise unwholesome condition, or where there is some doubt as to their places of destination. Bilge water can be pumped out, and the supply of drinking water changed.

In actual practice the port medical officer keeps a register of ports which he considers as "infected." This list is revised from time to time from various official and unofficial sources. There is scope for much discretion in adding the names of ports to such list. It frequently happens that a newspaper paragraph gives the first warning of cases of disease in a foreign port. Often such rumour is followed by an official denial, and the fact is only admitted when it is impossible any longer to conceal it.

This list is a confidential one for the use of the authority only, and should never be published. It, therefore, can in no way damage the interests of any port or its trade if its name be added thereto, while its inclusion may be a most valuable protective precaution.

The instructions to the boarding medical officers should be:—

(i.) To visit every vessel arriving from "foreign" ports, and to examine every case of sickness reported, and all the crew when any suspicion exists.

(ii.) In the cases of all vessels from ports mentioned on—

(*a*) The "infected" list, to board and medically examine every person on board, checking the number by means of the crew and passenger lists.

(*b*) To obtain from the master (or surgeon if one is carried) all records of sickness during the voyage, and examine the log-book as to entries relating thereto.

(*c*) To obtain an accurate list of all the names of those on board, their places of destination, and addresses at such places of destination.

(*d*) To forward notification of such names and addresses to the sanitary authorities concerned.

(iii.) Where vessels are found to be "infected" as a result of such medical inspection, to serve a certificate on the master to this effect, and proceed to carry out the terms of the Local Government Board, reporting all the details to the Board.

Such a system appears to combine the maximum of protection with the minimum of interference with trade, and it is found in practice that shipowners will do their utmost to assist the sanitary authority so long as they feel that their interests are studied as far as possible, with due regard to the protection of the public health.

One other point should be insisted upon, viz. the production of a "medical history" of the voyage, giving all facts connected with the health of those on board, and enumerating all cases of sickness, however trivial. Where a surgeon is carried this is a most valuable help and assistance, and where one has to rely upon a master it will often suggest what might be otherwise overlooked. From such a one facts only are required, and not opinions, for it is obviously absurd to expect to obtain a diagnosis of disease from any but a medical man.

Many of our leading shipowners have already adopted this system, and good results have been obtained, but it seems most desirable that it should be made compulsory on all.

Such a system as this involves considerable expenditure, but it should be looked upon as a system of insurance, and therefore regarded as a wise and prudent outlay. The question of dealing with cholera, yellow fever, and plague must be regarded as rather a commercial than a medical one, when one considers the enormous loss to a country which ensues from the paralysis of trade which invariably follows any outbreak of these dreaded diseases. While smallpox may destroy thousands and disfigure tens of thousands without any appreciable loss to a country, the occurrence of only a dozen cases of plague may inflict a loss of millions of pounds to the trading community.

Quartan.—Recurring on the fourth day; *e.g.* a form of intermittent fever in which a paroxysm occurs once in every three days, there being two days quite free from it. *See* MALARIA (*Clinical Manifestations, Quartan*).

Quarter-Evil. *See* ANTHRAX (*Synonyms*).

Quartipara.—A woman who has given birth four times, or one who is in her fourth pregnancy.

Quassia.—Quassiæ Lignum is the wood of *Picrasma excelsa*, a large tree of Jamaica. It contains *quassin*, an intensely bitter principle irritant to mucous membranes; and a volatile oil. There is no tannin present, so that quassia can be prescribed along with salts of iron. *Preparations* — 1. Infusum Quassiæ. *Dose*—½-1 ʒ. 2. Tinctura Quassiæ. *Dose*—½-1 ʒ. 3. Liquor Quassiæ Concentratus. *Dose*—½-1 ʒ.

Quassia is a simple bitter tonic much used in cases of anorexia and gastric inactivity. It has been employed during convalescence from malaria, on the supposition that it possesses antiperiodic properties. It is one of the best agents to use for the destruction of threadworms. After the bowel has been thoroughly washed out with a soap and water enema, about half a pint of the infusion is injected well into the rectum. A few such injections almost invariably kill the parasites.

Quebracho. — White quebracho bark (*Quebracho cortex*) is the bark of one of the Apocynaceæ, viz. *Aspidosperma quebracho*; it contains several alkaloids, such as *aspidospermin* ($C_{22}H_{30}N_2O_2$), *aspidospermatin* ($C_{22}H_{28}N_2O_2$), and

quebrachin ($C_{21}H_{26}N_2O_3$), which in small doses increase the fulness and frequency of the respiration, and in larger doses paralyse it; it is not now officinal, but it was formerly in the B.P., and there was a *tinctura quebracho* which was used in dyspnœa in doses of ½ to 1 fl. dr.

Quercin.—A bitter carbohydrate, $C_6H_6(OH)_6$, obtained from acorns.

Quercite.—A sweet carbohydrate, known as oak-sugar or acorn-sugar, having the formula $C_6H_7(OH)_5$.

Quercus Cortex.—Oak bark, obtained from *Quercus robur*, is not now official in the B.P.; it was formerly employed to prepare the *Decoctum Quercus*, which was employed (on account of its contained tannin) as an astringent.

Quickening.—The feeling of life, or stirrage, usually detected by the pregnant woman about the mid-term of gestation. *See* Pregnancy, Diagnosis (*Symptoms*).

Quicksilver. *See* Mercury; Trades, Dangerous (*Mercury*).

Quillaiæ Cortex.—Panama bark, soap bark; obtained from the tree *Quillaja saponaria*. Its chief constituent is a glucoside, *saponin*. *Preparation*—Tinctura Quillaiæ. *Dose*—½-1 ʒ. The tincture of quillaia might be used for emulsifying oils and other insoluble substances, but other more suitable agents are generally employed for this purpose. On account of the saponin present it has an expectorant action similar to that of senega, and has been recommended highly by some for bronchitis and other chest conditions.

Quincke's Disease. *See* Angeioneurotic Œdema.

Quincke's Lumbar Puncture. *See* Meningitis, Epidemic Cerebro-spinal (*Diagnosis*); Spine, Surgical Affections of (*Lumbar Puncture*).

Quincke's Method.—A mode of estimating the total quantity of blood. *See* Blood (*Methods of Clinical Examination, General*).

Quinidina. *See* Alkaloids (*Vegetable*); Pharmacology; Quinine; etc.

Quinine. *See also* Alkaloids; Amblyopia (*Toxic*); Colour Vision (*Acquired Colour Blindness, Causes*); Drug Eruptions (*Quinine*); Hæmoglobinuria (*Toxic*); Purpura (*Symptomatic, Toxic*); Tinnitus Aurium (*Treatment*).—An alkaloid obtained from the bark of one of the numerous species of the genus *Cinchona*. The official form is the Red Cinchona Bark—Cinchonæ Rubræ Cortex—which contains (in addition to *quinine*) *quinidine*, *cinchonine*, *cinchonidine*, and other substances of less importance. Good bark should contain between 5 and 6 per cent of alkaloids, of which at least half should consist of quinine and cinchonidine. Quinine is the most important of the group, and its physiological action is practically the same as that of the others. The *dose* of the bark is 3-15 grs.; and 30-120 grs. in malaria. *Preparations*—1. Extractum Cinchonæ Liquidum. Contains 5 per cent of alkaloids. *Dose*—5-15 m. 2. Tinctura Cinchonæ. Contains 1 per cent of alkaloids. *Dose*—½-1 ʒ. 3. Tinctura Cinchonæ Composita. Contains ½ per cent of alkaloids. *Dose*—½-1 ʒ. 4. Infusum Cinchonæ Acidum. Prepared with aromatic sulphuric acid, and is a solution of the sulphates of the alkaloids. *Dose*—½-1 ℥. These preparations must all be made from red bark.

Quinine, is official in the form of its salts, which may be prepared from any member of the cinchona group. I. *Quininæ Sulphas*. Slightly flexible, filiform, snow-white crystals, soluble 1 in 800 of water (giving it a fluorescent, bluish tinge), easily in acidulated water. *Dose*—1-5 grs. as a bitter; 5-20 grs. as an antiperiodic and antipyretic. *Preparations*—1. Tinctura Quininæ Ammoniata. *Dose*—½-1 ʒ. 2. Pilula Quininæ Sulphatis. *Dose*—2-8 grs. 3. Ferri et Quininæ Citras. *Dose*—5-10 grs. 4. Syrupus Ferri Phosphatis cum Quinina et Strychnina. ⅘ gr. in each drachm. *Dose*—½-1 ʒ. II. *Quininæ Hydrochloridum*. Soluble 1 in 35 of cold water. *Dose*—1-10 grs. *Preparations*—1. Tinctura Quininæ. *Dose*—½-1 ʒ. 2. Vinum Quininæ. *Dose*—½-1 ℥. III. *Quininæ Hydrochloridum Acidum*. A white crystalline powder soluble in less than its own weight of water. *Dose*—1-10 grs. There are also other non-official salts in use, including the *bisulphate*, the *hydrobromide*, the *chlorate*, the *citrate*, the *lactate*, the *phosphate*, the *salicylate*, and the *valerianate*. *Marburg's Tincture* (Tinctura Antiperiodica) has a high reputation in India for malaria. It contains aloes, rhubarb, camphor, and opium, along with many aromatics. As it is rather apt to purge it may be prescribed without aloes.

Quinine is a powerful antiseptic, but it is too expensive to be used for this purpose. Internally it is administered for three chief purposes—as an antiperiodic or antimalarial, as an antipyretic, and as a tonic. In malaria it is a specific, its action being directly poisonous to the plasmodium malariæ in the blood. The amount given varies greatly, and must be decided upon according to the indications from the state of the particular patient. Its administration should be preceded, if possible, by a cholagogue purge. It may be given in hourly doses or in single large doses so timed that they will begin to have effect about half an hour before the expected paroxysm. If the attack is imminent, it should be taken in solution. Even after the

onset of a paroxysm quinine should be administered, so as to destroy the spores which have been set free. In severe forms of the disease and in pernicious malarial fever quinine may be given hypodermically or even intravenously in large doses. The best salts for these purposes are the lactate and the acid hydrochloride. The hypodermic should be given into the buttock, and the intravenous injection into a vein of the leg in very dilute solution, injected slowly, so as to avoid the depressant effect on the heart. In neuralgia of malarial origin quinine acts very beneficially. Quinine is *the* prophylactic against malaria, and should be taken in doses of from 2 to 4 grs. three times daily by susceptible individuals in malarial districts. After suffering from malaria, the patient should continue to take quinine until a complete cure has been effected. Quinine is not the cause of blackwater fever, as has been supposed, but it should not be given in this disease unless the parasites are actually present in the blood. Quinine is not now largely used as an antipyretic. To obtain this effect to any marked degree large doses must be employed, and these are depressing. It is often prescribed empirically for influenza in comparatively small doses. It is employed as a bitter tonic in general debility, in anæmia, and in gastric inactivity. In some cases of neuralgia it has a good effect, best given perhaps in a single large dose. It increases the contractions of the uterus, and is helpful in uterine inertia during labour. It will not of itself produce abortion, and may safely be administered, when required, to pregnant women with or without the addition of opium or a bromide. It has been recommended for whooping-cough, in doses of $1\frac{1}{2}$ gr. three times daily for every year of the child's age. In the strength of 1 or 2 grs. to the ounce, it may be sprayed on the throat in colds of the head and in tonsillitis. High rectal injections of the strength of 1 in 3000 have proved of value in amœbic dysentery. For the general tonic effects the cinchona preparations are often prescribed, but for all other purposes the salts of quinine are to be preferred.

Quinism.—The condition produced by large doses of quinine, or by ordinary doses in specially susceptible individuals; cinchonism; the symptoms are ringing in the ears, headache, deafness, etc. *See* QUININE.

Quinoline.—An alkaloid (C_9H_7N) related both to benzene and to pyridine, and obtained from coal-tar or from the destructive distillation of quinine.

Quinone.—When quinic acid ($C_7H_{12}O_6$) is oxidised, quinone ($C_6H_4O_2$) is formed. *See* HYDROQUINONE.

Quinosol. *See* CHINOSOL.

Quinquaud's Disease.—An affection of the hair follicles of the skin with cicatrisation; tinea decalvans.

Quinsy. *See* TONSILS, DISEASES OF (*Acute Tonsillitis*); DIPHTHERIA (*Diagnosis*).

Quintan.—Recurring after three days interval, viz. on the first, fifth, ninth, thirteenth days, and so on; *e.g.* quintan ague. *See* MALARIA.

Quintipara.—A woman who has given birth five times, or who is pregnant for the fifth time.

Quintuplets.—Five children at a birth. *See* PREGNANCY, MULTIPLE (*Varieties*).

Quotidian.—Recurring once in twenty-four hours; *e.g.* ague, which has a paroxysm daily. *See* MALARIA.

Quotient.—The result of division, *e.g.* the *blood quotient* (colour index) is the proportionate amount of hæmoglobin contained in each red blood corpuscle, and this is obtained by dividing the quantity of hæmoglobin in the blood by the number of erythrocytes.

Qushuf. *See* PELLAGRA (*Synonyms, Egypt*).

R.—The contraction for *recipe* in prescription-writing. *See* PRESCRIBING.

Rabbit Soup. *See* INVALID FEEDING (*Convalescence*).

Rabies.

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SYN.—Hydrophobia.

RABIES is a virulent disease transmissible by inoculation, and contracted by man from the bite of a mad animal.

HISTORY.—Although rabies is without doubt a disease of ancient times, it was not recognised by the physicians of olden days. Aristotle writes about the rabies of dogs, but he firmly believed man to be exempt from it. The Alexandrian doctors were the first to record reliable facts about human rabies.

During the Middle Ages, and indeed until the

close of last century, rabies was regarded as a mysterious neurosis, the most astounding hypotheses being formulated as to its nature; only one true fact was admitted, namely, the transmission of rabies to man by the bite of an animal.

In the last century the experiments of Majendie, Breschet, Renault, Hertwig, Benley, etc., clearly demonstrate the condition and development of this scourge. Our present more accurate knowledge of the pathology of this disease, and the solutions of the problems of prophylaxis and vaccination, as already practised by Galtier on *animals*, is mainly due to the exertions of Pasteur and his pupils, who have been equally successful in the cure of *man*.

Etiology.—Rabies is found in every quarter of the globe, in both hot and cold regions. In Europe the disease is most prevalent in France, Belgium, and Russia.

In France, during the year 1892, there were no fewer than 2169 rabic dogs. In the department of the Seine the malady is practically endemic. In Belgium during the same year there were 83 cases of rabies in dogs; in Germany the cases were much fewer, only 387 being reported in 1892; while England enjoys a still greater immunity, statistics proving 79 cases in 1891, and only 38 in 1892. From year to year, of course, the number of cases varies—an increase of rabies invariably following any relaxing of the vigilance of the police in enforcing sanitary regulations.

The influence of the seasons is not nearly so important as was formerly believed to be the case.

Rabies sometimes manifests itself in an epizoötic form; such was the case at Peron in 1803, where the ravages caused by rabies are to this day mentioned with horror.

Some countries, for example Australia, enjoy almost complete immunity from rabies,—thanks, no doubt, to the vigilant inspection of vessels on arrival at ports. Other islands hitherto immune have suffered from the pest, owing to the admission of ownerless dogs; for example, Mauritius in 1813, Malta in 1847, and Madeira in 1892.

Rabies in man being always contracted from animals, the etiology of the condition in animals must therefore first be considered.

All mammalia are liable to rabies, the different species differing in regard to degree, frequency, and severity. Domestic animals, on account of the life they lead, are most susceptible; rabies appears most frequently in dogs, cats more rarely suffer. Among the wild carnivora the wolf is frequently attacked, also the fox, hyena, and jackal. Oxen are the most susceptible of the herbivora; next in order come sheep, goats, and horses. Rabies in pigs is not so common. It is doubtful whether birds can be attacked by rabies.

In all the different animal species males suffer from rabies more frequently than females. Le Blance makes the proportion 14 to 1 in the canine races.

It is now comparatively easy to estimate the extent of rabies in man, thanks to the accurate statistics of the Pasteur Institute. In France, from 1800 to 1900 individuals are yearly bitten by animals and undergo the Pasteur treatment. One thousand, at least, have been bitten by certified rabic dogs. The greater number of the cases of bites from rabic dogs occur in the department of the Seine, where there are at least 47 individuals bitten to each 100,000 of the population.

Men, as a rule, are more frequently and severely bitten than women; children do not suffer so often as adults. These facts are due probably to the different modes of life, clothing, etc.

The bites of rabic animals only communicate rabies in about a third of the cases in animals, and the same may probably hold good as regards man. As it is impossible to reckon all bites, this is difficult to state quite accurately; the proportion, roughly speaking, may be taken as 16 to 19 cases of rabies against 100 bites.

All bites are not equally venomous. Wolf bites more frequently communicate rabies than dog bites, not because the poison is more virulent in the wolf, but because he bites deeper and more than once. The bites of herbivorous animals are not deep—they tear rather than bite—and are therefore less serious.

Bites on exposed flesh are naturally most serious. If the clothes intervene the saliva may be partially wiped off the teeth in passing through to the flesh. Bites on the face are the most fatal, the mortality is 88 per 100; on the hands, 67 per 100; and on the lower limbs, 21 per 100. The vehicle for the virus is the animal's saliva, and the teeth carry it into the flesh. The saliva merely spread on the surface of the skin does not cause rabies unless there is a scratch or wound.

The theories of contagion in the air, or by the perspiration, or by the spermatic fluid, are not credited now; even infection by the blood of rabic animals is doubted, as is also infection through administration of milk or meat.

Symptoms.—I. *Period of Incubation.*—The period of incubation, running from the moment of the bite till the appearance of the known symptoms of rabies, is very variable. The ancients considered incubation to last about forty days. Recent statistics of Brouardel, Proust, Bauer, and Meintrier allow approximately two months after inoculation, sometimes three months, and in very rare cases six months.

The extreme limit is difficult to fix. Bouley has seen cases of incubation lasting only seven days. De Beurmenn Valentin had cases run-

ning on from sixteen to eighteen months. Chantemem knew a case of a patient who had been treated with preventive vaccine, where the incubation lasted twenty-seven months. Certain conditions tend to alter the periods of incubation. It is shorter if there are many bites, and if the face is bitten probably forty-eight days instead of sixty-nine days. Incubation is generally shorter in the case of children, owing to their faces very often being bitten; fifty-seven days is the average under twenty years of age, and sixty-eight days above that age.

All excesses, over-eating or drinking, are said to hasten the attacks, but this is a doubtful assertion. All doctors agree in trying to discover during this period of incubation *premonitory symptoms* of the approaching rabies. Some have thought that the scar has a special aspect, it cicatrises slowly, and the wound remains swollen.

An early theory was that a little worm grew under the tongues of dogs, and this brought on rabies. At a later period doctors thought they discovered under the tongue of the bitten person certain vesicles or lesions which were an undoubted test of rabies. Needless to say, such theories have no foundation in fact.

The only symptoms worthy of notice are functional symptoms which often appear, such as formication, pains starting from the wound and rising to the head and heart, cold shiverings of the wounded limb, with anæsthesia and hyperæsthesia, local paralysis, fibrillary tremors, etc.

When it is known that the hydrophobic virus is pursuing its course through the nerves these phenomena are accounted for, and the patient should then undergo very rigorous treatment.

II. *Prodromic Period.*—The prodromic period is characterised by mental and bulbar disorders. A psychic depression comes on with extreme melancholia. The patient seeks to be alone, and is haunted by fear of the dreaded malady, and disturbed by nightmares and insomnia. He suffers from general debility, and shuns his ordinary routine of life; cephalalgia and neuralgia in the seat of the wound are sometimes present. Many persons declare they see apparitions that urge them to commit suicide. During this phase there is no excitement or extraordinary quickening of the senses, or desire for inordinate movement. Later, the breathing is disturbed and incessantly interrupted by deep sighs, which contract the diaphragm to its utmost limit; the other respiratory muscles come into play, and the sufferer experiences a sensation of præcordial agony and thoracic constriction or spasm; the shoulders are pushed up. The duration of the phase is difficult to determine; sometimes it may last only two or three days, sometimes several weeks, especially if there has been a complication of nervous disorders at the period of incubation.

III. *Period of Excitement or Hydrophobia.*—This period is characterised by excessive irritability throughout the whole nervous system, specially the region of the medulla. Exaggeration of the reflexes explains the occurrence of the spasms, the most characteristic of all being the *hydrophobic spasm.*

The breathing is interrupted by heavy sighs and spasms as if one had suddenly been plunged into a cold bath. Cardiac oppression and dyspnœa develop rapidly. Spasms in the pharynx and larynx cause special difficulty in deglutition and are the true hydrophobia. When the patient desires to drink, the moment he raises the glass to his lips his face is convulsed, his eyes fixed, his body trembles, and it is impossible for him to swallow one drop of liquid. After resting a little, being still tormented with thirst, he again tries to drink; the same phenomena recur, and he cannot quench his thirst. The mere sight of water or sound of liquid poured into a glass, the mere thinking of it, or even trying to swallow solids, brings on the spasms. Occasionally certain things are tolerated, such as lumps of ice, and possibly the sufferer may be able to drink in the dark. All the reflexes of the skin, tendons, and oculo-pupillaries are exaggerated; the trembling of the muscles excites myodemia.

Any afferent stimulant, *i.e.* a sound, a breath of air, etc., on the surface of the skin induces convulsions and spasmodic breathing. The eyes are wildly bright, the pupils from time to time either abnormally dilated or contracted. Bright light seems to rouse the patient to paroxysms of fury; he shuns it, and seeks to hide in the dark.

The senses of hearing and smell are abnormally quickened; some smells bring on acute spasms, and the patient suffers from hallucinations of sight and hearing. These convulsions increase in frequency and intensity, and develop the real hydrophobic spasm. At times the spasms are convulsive; at times acute mania is developed. The precardiac agitation increases; the patient appears on the point of suffocation; his body quivers with shocks, as in an epileptic fit, or becomes rigid, as in tetanus. The attacks may at first be short and at intervals; they gradually increase in duration and frequency, lasting from fifteen to twenty minutes. The patient is frequently attacked by frenzies of passion; he screams and flings himself about, biting and tearing the bedclothes, and knocking himself against the wall. The voice becomes hoarse and he utters a barking sound, the eyes roll, the saliva dribbles from the mouth; his aspect is most alarming. In spite of these paroxysms the unhappy sufferer vainly tries to avoid biting those near him, he dreads hurting any one.

Consciousness returns during the intervals between the attacks, and the patient is over-

whelmed with intense grief and melancholy, which presents a striking contrast to his former mad fury. He rarely sleeps, starts up dreaming, the mouth hangs open, the saliva cannot be swallowed; fearing to injure his attendants, the patient begs them to put him out of his misery and end his life. The face and limbs are bathed in sweat, there is obstinate constipation, often vomiting of bile and blood. The urine is seldom passed, and is found to contain sugar and albumin. Samson and Chippingdale have observed hæmoglobinuria and aglobulia. Genital disorders frequently supervene; the sufferer has emissions accompanied with great pain, and forcing cries of agony.

Fever increases as the prodromatic phase progresses; it abates slightly in the mornings. As death approaches the temperature rises very high, as much as 43°. Even after death it may still continue high.

The pulse is rapid and intermittent.

Death may result from asphyxia during the course of such an attack.

The period of excitement may last one or two days; death invariably results from asphyxia or syncope.

IV. *Paralytic Period.*—This period only lasts a few hours. The sufferer loses all consciousness and falls into a state of collapse. In very rare cases consciousness may return at the point of death.

Forms.—The ordinary form of rabies is characterised by a complete disorganisation of the whole nervous system. More rarely the furious and preliminary stages are absent and the rabies is manifested by paralysis from the outset; the paralysis appears, accompanied with acute local pains, mental depression, and other sensory troubles. The sufferer moves the bitten limb with difficulty, there is paresis with loss of co-ordination present; and later the paralysis becomes total. At first limited to a monoplegia or paraplegia, it extends, accompanied by severe pain, to other regions of the body; tongue, face, eyes, bladder, rectum, and intestinal muscles are all paralysed, and there is constipation and retention of urine. Lastly, though not invariably, the vital centres in the medulla are affected, breathing disorders set in, and with them true hydrophobia. The paralysis usually commences in the bitten limb; occasionally it may begin in the opposite side. The paralysis comes on at a late stage, after the usual symptoms have set in.

Finally, where subjects have been inoculated with Pasteur's vaccine, rabies appears in a more modified degree, limited to the prodromic phenomena, as observed by Chautemann, Roux, and Laveran. Rabies does not seem to depend on the kind of bite. Gamaleia contends, however, that the worst bites bring on paralytic rabies; on the other hand, Di Vestea and Zagari maintain that ordinary rabies results from bites on the head, and paralytic rabies or "dumb rabies" from bites on the lower limbs.

Animal Rabies.—All the animal species suffer from the two forms of human rabies, namely, hydrophobia and dumb rabies.

With dogs furious rabies is the most common, and exhibits itself in much the same way as in the human race. Depression, depraved appetite, hallucinations, fear of water, changed bark, dysphagia without hydrophobia, and fever. Presently the animal is seized with a frenzied restlessness, a determination to bite any thing or person he meets. Death follows paralytic collapse.

Dumb rabies is exhibited by a slow paralysis which first centres in the muscles of the jaw. Death results in three or four days.

Progress—Duration—Termination—Prognosis.—The course taken by rabies is progressive, rapid, and fatal. The bulbar phenomena precede the cortical phenomena; the rabic virus mainly attacks the nerve centres.

The duration is difficult to determine on account of variability in the prodromic period; but from the first symptoms to the development of true hydrophobia the course is rapid, and death takes place in three or four days, in some cases even on the first day. Paralysis is slower, though not less fatal, death ensuing on the seventh or eighth day.

Rabies is almost always fatal, and death is due to asphyxia or syncope. Death may also result from traumatism or suicide during the frenzied period. Veterinary surgeons have recorded cases of recovery from rabies in animals; possibly these were mild cases of slight inoculation on an anterior part (Hagges). Laveran, Roux, and Chantemesse have recorded cases of rabies being cured when the patient underwent treatment at the Pasteur Institute. Novi and Poppi knew a case of a patient suffering from rabies, who by the twentieth day of the curative treatment was almost cured. It is thus evident that rabies is not necessarily absolutely fatal.

Diagnosis.—The diagnosis of human rabies as a rule is simple, though there are certain diseases which may simulate rabies.

The *imaginary hydrophobia* of a bitten person is excited by suggestion in a neurotic subject. If the patient has some knowledge of the symptoms of human rabies the diagnosis may be difficult. The patient may also imitate canine rabies. If it can be proved that the suspected dog was not mad, it may be well to assume, in the absence of a period of incubation, that the symptoms are merely hysterical stigmata simulating rabies.

Tetanus has a much shorter period of incubation, is always accompanied by contractions, especially a persistent trismus, but it never develops into real hydrophobia.

Certain *poisons*, such as stramonium and

belladonna, exhibit some symptoms akin to rabies.

In *delirium tremens*, hallucinations and delirium usher in the attack; whereas in rabies, spasms and respiratory disorders predominate from the outset.

Paralytic rabies is a form of acute paralysis, whose etiology is the only guide to its correct diagnosis.

It is generally a simple matter to recognise human rabies; it is a more difficult task to foresee and determine rabies in the dog, but this is a matter of the gravest importance; and this diagnosis is absolutely necessary in order to judge of the course to be pursued in treating the bitten man or animal.

Rabies is not always easy to recognise in the living animal. It is neccssary to shut up the suspected dog and keep him under vigilant observation for at least forty-eight hours. By killing the dog too soon the risk is run of not obtaining from the autopsy definite or satisfactory indications of the disease.

In cases where the presumption of rabies justifies it, the animal must be slaughtered, and a diagnosis made by a post-mortem examination. The same examination should be made by the veterinary surgeon, even if the animal has been slaughtered before he sees it.

The presence of foreign substances in the stomach and intestines, the absence of food in the alimentary canal, dryness of and accretions on the mucous membrane, the presence of glycosuria, are indications of the disease, but not conclusive ones.

The histological examination of the nerve centres furnishes still more convincing proofs. Babés studied a portion of the medulla of a dog, and searched for the rabic nodules (*see* p. 460) formed on the nerve tissues and surrounded by areas of small cell infiltration. Their presence convinced him that the case was one of true rabies. Van Gehuchten studied the ganglion of the pneumogastric nerve, and asserted that in rabic animals there were specific alterations of the nerve cells. Piliet and Vallet admit that the lesions described by Van Gehuchten are peculiar to rabies, but they appertain to an advanced stage of the disease, and are never found in the animals slaughtered in the early stages of the disease. Marinisco considers that the nerve lesions noted by Babés and Van Gehuchten are unreliable symptoms, being neither specific nor invariable.

To obtain a correct diagnosis, we must have recourse to bacteriological proof, a portion of the medulla of the suspected animal being aseptically inoculated into the eye of several susceptible animals, such as dogs or rabbits. Unfortunately this diagnosis is a very lengthy process, satisfactory results seldom being obtained before thirty or forty days, indeed often not till the ninetieth day. Therefore persons who have been bitten should at once be sent to the Pasteur Institute without waiting for this tardy result.

Pathological Anatomy.—For a long time the anatomical lesions of rabies were entirely unobserved. By degrees physicians began to describe certain vascular lesions in conjunction with small cell infiltrations of the nerve centres, and in consequence rabies was treated as an encephalic myelitis. Since 1890 histologists have observed various microscopic lesions, the exact nature of which has not yet been explained.

At the autopsy of a man or animal who has died of rabies, the cerebral and spinal meninges are invariably found to be greatly congested and œdematous, with hæmorrhages at the vein centres. The adjacent nerve tissue presents a congested appearance, with small hæmorrhagic spots. These lesions are most numerous in the region of the medulla, the basal ganglia, spinal cord, and anterior and posterior nerve roots.

Microscopical examination reveals alterations in the blood-vessels; they are surrounded by small cell infiltrations, which give them a moniliform appearance. These nodules consist of clusters of round perivascular cells. The veins appear to have undergone a hyaline degeneration (Kolesnikoff). There is also small cell infiltration and a multiplication of the fixed cells, the transparency of the veins becoming obscured by a thrombus or by the accumulation of leucocytes. The diseased veins are liable to burst. Benedict found miliary aneurysms; Schaffer and Babés observed capillary hæmorrhages. The nerve cells of the cord and the medulla undergo marked alterations. Schaffer and Poppof studied the pigmentary atrophy of the cells in two cases of human rabies. Babés, Golgi, Nagy, Marinisco, etc., describe these chromatic phenomena, changes in the colouring matter and in the protoplasmic cells, actual disappearance of the chromatic elements, vascular degeneration, loss of the cellular prolongment, and modification and disappearance of the nucleus. Babés follows up Kolesnikoff and Coats in describing the relations between the cells; tells how they are invaded by small round cells and hyaline corpuscles. He lays special stress on the pericellular miliary foci, which he styles rabic tubercles, and deems an invariable accompaniment of rabies.

Van Gehuchten and Nélis studied the lesions of the cerebro-spinal ganglia, which they in their turn consider specially characteristic of rabies. Numerous nerve cells disappear, and are replaced by clusters of small round cells. The remaining cells present various lesions of the protoplasm and nucleus; at times even the nerve cells of the ganglion entirely disappear. These lesions attain their maximum in the nodose ganglion of the pneumogastric. Polaillon and Nerven had already noticed analogous lesions in the Gasserian ganglion. The most noteworthy

changes are (according to Schaffer) found in the medullary segments, which have a nerve connection with the seat of the bite. In the white substance the axis cylinders become enlarged, either denuded or contrariwise, surrounded by a thick veil of myeline. The peripheral parts of the nerve, especially the nerves of the bitten limb, undergo degeneration.

A study of the lesions of the other organs is equally interesting and characteristic. Pharyngitis, congestion of the salivary glands, gastric congestion, hepatic congestion, etc., are all noticeable. An examination of the stomach proves of use in dogs: the presence of unusual objects in the viscera, of whatever kind, is most essential in a retrospective diagnosis of rabies.

The bronchial mucous membrane and the lungs are congested, the kidneys are often the seat of nephritis. The lymphatic glands are enlarged; the blood grows diffluent and dark in colour, as it does in grave infectious fevers; hæmorrhages occur in the different organs.

Bacteriology.—*Experimental Rabies.*—The experimental study of rabies has been most exhaustively investigated by Pasteur and his students. Though it is still impossible to state definitely the precise nature of the rabic virus, their able researches have found out the seat of the virus in the organism, its mode of introduction and propagation, and the best means by which its virulent activity may be modified. This study brings us to the practical application of the Pasteur vaccine.

Seat of the Virus in the Organism.—It was long supposed that the saliva of rabic dogs and human beings was virulent, and Grunner and Salm Reiferschidt in 1813 furnished proofs of this. Novard and Roux later have proved that this is only supposition, and show that virus in the saliva is capricious and variable, and greatly depends on the point of inoculation. In the case of a dog inoculated in the eye, the saliva became virulent *three days before* the appearance of the nervous phenomena, and in a dog inoculated in the neck the virus became virulent *thirty hours before* the animal developed rabic symptoms. The virulence of the saliva does not proceed from a dilution with the bronchial mucus as P. Bert believed. The experiments of Hartwig, Galtier, and Bardach are proof that the salivary glands themselves contain the virus. Galtier, Breschet, Renault, P. Bert, etc., all agree that the virus does not exist in the blood, the lymph, or the muscular tissue. In rare cases the milk has been found to be infected (Perroncito, Carita, and Nocard). The poison also may occasionally infect the glands and various secretions of the body, *e.g.* pancreatic fluid, spermatic fluid, urine, etc.

The nervous system contains the virus with marked and unfailing certainty. It is always found in the medulla, in the spinal cord and brain. The white substance contains an equally virulent virus as the grey substance. Pasteur proved that for the purpose of inoculation the medulla, treated aseptically, may be used as a far more reliable inoculant than the saliva. The virus is also found in the peripheral nerves, but not so invariably (Roux).

Modifications of the Virus by Physical, Chemical, and Biological Agents.—In the cadaver the rabic virus has been found to resist putrefaction for forty-five to fifty days (Galtier). Its virulence progressively diminishes (Russo, Travali, and Brancaleone).

Roux recommends glycerine as the best agent in which to preserve the rabic medulla; the bulb may be kept in glycerine for four weeks without any sensible diminution of its virulence.

Heat has a powerful effect on the rabic virus, which is rapidly destroyed at 60° (Roux), in one hour at 90°, and in twenty-four hours at 49° (Celli). Cold, on the other hand, has little or no effect; the medulla of a rabic rabbit preserved its virulence for ten months kept at a temperature of 10° to 27° (Celli). The freezing of 7° to 8° atmosphere has no effect. The action of light can destroy the virus in forty hours at 37° (Celli).

Antiseptics quickly destroy the virus (Celli).

Pasteur makes use of the combined action of the air, of desiccation, and of light in diminishing the strength and ultimately destroying the virus, and also in obtaining spinal cords possessing varying degrees of virulence. The spinal cord of a rabbit exposed to a drying air at 23° loses its virulence in six days.

The activity of the rabic virus may be modified at will by its passage through several animals. The virus of dogs whose mode of existence has already inoculated them with "the virus of the streets" is less deadly. The virus from one of them inoculated under the dura mater will give rabies after an incubation of fifty days.

An increase of the virulence is obtained by successive passages through rabbits, the inoculation each time being made from the medulla of the last rabbit. At the 133rd passage the duration of the incubation is seven days; at the 178th it is six days (Pasteur). The "virus of passage" is a "fixed virus." It retains its strength in passing through a dog, and always kills the rabbit with the same incubation period. The rabbit is thus a reactive agent on account of the purity and strength of the rabies, which in its case assumes the paralytic form, the paralysis commencing in the posterior parts, and is preceded by increased rapidity of breathing, by polyuria, a loss of weight, and rise of temperature. The "fixed virus" is more rapidly obtained by passage through young animals (Hogyes). An attenuation of virulence is obtained by passage through the monkey.

Inoculation with Virus.—All parts are not equally favourable for inoculation. The surest and most rapid entrance is inoculation in the seventh nerve; its results never fail. Pasteur has also trephined the cranium and injected the virulent liquid under the dura mater with equal success.

The same holds good of inoculation into the subcutaneous lesser nerves, by tempering a section with one drop of virus (Di Vestea and Zagari).

Intra-muscular inoculation has much the same measure of success (Helman).

Inoculation by scarification of the skin succeeds only in one-third of the cases, and has besides a very variable incubation. It is only resorted to if the virus is impure.

Inoculation of the subcutaneous cellular tissue of the serous or mucous membranes cannot be relied upon. Bardach and Nocard are very doubtful whether injections of rabic spinal cords into the mucous membranes of animals is effective. Galtier also obtained very doubtful results from painting the mucous membrane with virus, so much depends on the condition of the mucus. Inoculation into the blood results, according to circumstances, in either rabies or protective vaccination. In treating dogs, an injection of rabic saliva only infects them with rabies uncertainly, whereas the injection of an emulsion of the medulla always results in rabies (Roux). With regard to ruminants Galtier asserts that venous inoculation is vaccinative, not infectant.

Intra-uterine transmission of rabies is made by the blood-vessels; Perroncito, Carita, and Camillac and Gibier all agree as to this fact.

Mode of Propagation in the Organism.—Jaccoud had already surmised that the rabic virus is propagated through the nerves. Experiments by Di Vestea and Zagari have proved that after inoculation under the dura mater of the brain the toxins first appear in the medulla, proceeding thence to the lumbar enlargement. Again, if the sciatic nerve is inoculated, the lumbar enlargement becomes virulent before the medulla. Lastly, if the nerve is excised below the inoculated point, and the peripheric end cauterised, the infection is either abortive or checked for the time. In the same manner if, after inoculation of the sciatic nerve, the spinal cord is divided below the origin of the nerve, all the inferior segments of the cord and the nerves which have been divided become virulent, but the superior segment remains normal.

These facts have been confirmed by the experiments of Roux, who has found that only the nerves nearest the bite are virulent.

This accounts for the gravity and rapidity of the virulence when the bites are on the face, a region full of nerves, and nearer the medulla than the limbs.

As formerly mentioned, the virus may also be transmitted through the blood-vessels into the body.

Nature of the Virus.—The active agent of rabic infection is most probably a living organism. The progress of the disease, its inoculability, the resistance of the virus to physical and chemical agents, all point to microbes as the causal agent (Roux). The loss of virulence in filtered liquids, although they can be preserved and propagated by the filter, betrays the presence of microbes, although the particular microbe has not yet been isolated.

Immunisation.—In 1881 Pasteur, in the course of his researches, ascertained that certain dogs inoculated with non-fatal rabic virus were thus vaccinated against subsequent inoculations. His aim then was to procure this immunisation or vaccination by every effort in his power.

By passage of the virus a certain number of times through monkeys, Pasteur in 1884 was able to obtain a virus of diminished virulence. Progressive inoculations of this weakened virus were found to immunise dogs against the fixed virus, inoculated by trephining.

The following year Pasteur and his colleagues, Chamberland and Roux, discovered a new method of attenuating the virus by desiccation, more unfailingly certain than the other in its results; this method is still in vogue.

Subcutaneous injections of progressive virus immunise the dog against the fixed virus, even if a virulent inoculation precedes the vaccinal inoculation. These attenuated viri are obtained by suspending the rabic spinal cord of rabbits in a flask containing potash, kept in a temperature of 23°. By the sixth day the cord has lost all virulence. To prepare the cord for use, a piece 3 millimetres long is dissolved in 1 centimetre cube of sterilised bouillon.

The virus can also be attenuated by diluting an emulsion of rabic tissue in saline solution (Hogyes). Babés dilutes the cord tissue in the serum of an immunised dog. These viri are usually inoculated under the skin; Galtier also affirms that ruminants may be inoculated by the blood-vessels, but this process is not infallible. Immunity may be obtained very speedily. Thus it is possible to inoculate dogs, and in eighteen hours they can resist inoculation of the fixed virus under the dura mater. Ordinarily, immunity cannot be obtained before the tenth day.

Immunity renders the subjects capable of sustaining considerable doses of virus by inoculation. An operator has been able without difficulty to effect 209 inoculations in six months.

The immunity lasts one year in 21 cases out of 100 dogs so treated; two years, 33 per 100; five years or more with others (Pasteur).

The production of this immunised medium is not easily explained. Desiccation does not seem to act altogether as a diminisher of the

virulence of the rabic marrow, for in cases where rabbits have died from inoculation with the dried marrow their medulla still contains fixed virus. Immunity is not then obtained by inoculation with attenuated virus. Possibly the attenuated virus has only an impoverishing effect on the virus contained in the cord (Hogyes).

Pasteur has a theory that the immunising agent points to the presence of an antitoxin matter associated with the rabic microbe. The microbe keeps its virulence intact in all the desiccated cords, but there destroys itself more quickly than the vaccinal matter.

Vaccination with soluble substances elaborated from the rabic microbes has also been tried unsuccessfully. Babés and Cerchez, with moderate success, have employed blood of vaccinated animals as an agent. According to Nocard the vaccinated serum destroys the rabic virus when it is in contact with it, but it does not succeed in preventing its evolution.

Treatment.—*Prophylactic.*—The most efficient prophylactic measures for checking the spread of rabies are sanitary regulations properly enforced. It is essential that all cases of known or suspected rabies be at once notified to the authorities, who have power to slaughter all animals suspected of having been bitten. The total destruction of all the contaminated animals is almost impossible owing to the vast number of ownerless dogs. This difficulty has been met and considerably modified in countries where there is a sufficiently high dog-tax. The Veterinary Congress at Vienna in 1869 fully recognised this. The payment of the tax would be considerably aided if all dogs were obliged to wear a tax medal. Bavaria is a striking example of vigilant police surveillance. In 1873 there were 821 cases of rabies in Bavaria; the numbers fell to 69 cases in 1881, and 6 cases in 1884. The enforcement of police regulations has banished rabies from the Grand Duchy of Baden, Holland, and Sweden.

Curative Measures—(1) *After the Bite.*—When local treatment can be immediately applied it must not be neglected, always remembering that this treatment is very insufficient, and should not inspire a false security.

Immediately after the bite the wound must be attended to, first by tying a ligature on the limb above the bitten part, and then cauterising the wound deeply with a red-hot iron. If the teeth have not penetrated beyond the sound surface of the skin it is sufficient to bathe the infected parts with an antiseptic. Inoculation is always necessary, and ought to be done as soon as possible after the bite. According to the old method the treatment lasted ten days; an inoculation was made each day, commencing with the fourteenth day's marrow, and leading on successively to the fifth. At the present time this method is extensively employed, beginning with the fourteenth day's marrow and ending with the third. In serious cases the different degrees of marrow are used more rapidly, arriving on the third day to marrow of the sixth strength, and the fourth day to virulent marrow. The duration of the treatment varies, according to the gravity of the case, from fifteen to twenty-two days. If any pain is still felt at the seat of the bite a fresh series of inoculation is begun, which frequently may check the development of rabies. The inoculations are not very painful, and are only followed by a general local reaction, sometimes by erythema and itching. In order properly to estimate the results of the inoculation treatment we must look at the statistics of the Pasteur Institute. In 1896 the number of persons treated was 1308, of whom only four died. This gives a mortality of 0·30 per 100. When this mortality is compared with the mortality of non-treated rabies, which is 16 per cent, the immense benefit of the Pasteur treatment can be better appreciated. The statistics of the Pasteur Institutes in France and other countries agree with those of Paris. Nevertheless there are many criticisms and objections made to rabic inoculation. Indeed, it is accused of being inefficient and dangerous! The foregoing statistics conclusively answer the first objection, and Vulpion and Brouardel have replied to the second. They maintain and prove that of the inoculated cases which do not recover death is due to "the rabies of the streets," not to the "rabies of the laboratories," as Peter terms it.

(2) *After the Appearance of Rabies.*—The Pasteur treatment is ineffectual after the rabies is pronounced, unless it happen that the rabies come on during the course of the treatment—then there may still be hope. Noir and Poppi have saved cases of paralytic rabies, appearing about the twentieth day of treatment, by substituting intravenous injections for the subcutaneous. Other remedies are merely palliative. Large doses of morphia, chloral, chloroform, or bromide of potassium may be administered to soothe the paroxysms. The injection of artificial serum or continuous currents along the spine may be tried with good effect (Schivardi). Finally, it is essential to avoid all excitement, and keep the sufferer in a darkened room, avoiding light, noise, draughts, or anything likely to excite his extreme nervous susceptibility.

Race. *See* Cholera, Epidemic (*Etiology, Racial Relations*); Generation, Female Organs of (*Pelvic Differences due to Race*); Obesity (*Etiology, Race*).

Racemose.—Branched like a cluster of grapes (Lat. *racemus*, a cluster); *e.g.* a *racemose*

gland (*see* PHYSIOLOGY, TISSUES, *Secreting Epithelium*), *racemose aneurysm* (aneurysm by anastomosis).

Rachi-.—In compound words *rachi-* (Gr. ῥάχις, the backbone) means relating to the spine; *e.g. rachiagra* (gouty pain in the back), *rachialgia* (neuralgic pain in the back), *rachiocampsis* (spinal curvature), *rachioplegia* (spinal paralysis), and *rachiasmus* (spasm of the posterior cervical muscles).

Rachicele.—The protrusion of the contents of the spinal canal through an opening in the walls of the canal. *See* SPINA BIFIDA.

Rachicocainisation.—The production of anæsthesia by lumbar puncture, the anæsthetic (cocaine) being injected into the subarachnoid space. *See* SPINE, SURGICAL AFFECTIONS OF (*Spinal Cocainisation*).

Rachiotomy.—The division of the spine of the fœtus to permit of its extraction in difficult labours, *e.g.* in impacted shoulder cases.

Rachischisis or Rhachischisis.—Spina bifida (*q.v.*); the whole spine may be open (*holorhachischisis*) or only a part of it, such as in the cervical or lumbar regions (*merorhachischisis*).

Rachitis. *See* RICKETS; DEFORMITIES (*Genu Valgum*, etc.); SCURVY, INFANTILE; etc.

Rachitome.—An instrument for dividing or opening into the spine in rachiotomy.

Radesyge. *See* VENEREAL DISEASE (*Allied Diseases*).

Radial.—In Anatomy *radial* means relating to the radius (bone of the forearm) or to that aspect of the arm where the radius is situated, *e.g. radial* artery, nerve, pulse, or vein.

Radiator. *See* VENTILATION AND WARMING (*Heating by Steam*, etc.).

Radical or Radicle.—In Chemistry a radical (*radicle*, since 1862) is "an element or atom (*simple radical*) or a group of these (*compound radical*), forming the base of a compound and remaining unaltered during the ordinary chemical reactions to which this is liable" (Murray's *New English Dictionary*); in Surgery the term means thorough, touching what is essential, or going to the root of anything, *e.g.* a radical operation, a radical cure, etc.

Radio-.—In compound words *radio-* means relating to the radius (bone of the arm), or to radium (the metal), or to radiant energy; *e.g. radio-carpal* (relating to the radius and to the carpus), *radiodermatitis* (inflammation of the skin due to the action of the Röntgen rays), and *radiotherapy* (treatment of disease by radiant energy).

Radiography. — Photography, or the impression of objects on sensitised plates by the Röntgen rays, or skiagraphy, the result being a radiogram or skiagram. *See* X-RAYS. *See also* EYEBALL, INJURIES OF (*Foreign Bodies, Removal*); KIDNEY, SURGICAL AFFECTIONS OF (*Stone, Diagnosis of Renal Calculi*); LUNG, TUBERCULOSIS OF (*Diagnosis, Röntgen-ray Illumination*); MEDIASTINUM (*Growths, Diagnosis*); ŒSOPHAGUS, INJURIES AND DISEASES (*Foreign Bodies, Examination by Radiography*).

Radioscopy.—The examination of parts of the body or objects by means of the Röntgen rays. *See* X-RAYS.

Radium.—Radium, the remarkable properties of which have excited so much interest throughout the scientific world, was discovered by M. and Mme. Curie in 1898. The discovery was led up to by a number of different investigations extending back to the middle of the nineteenth century, when Geissler showed that an electric discharge through a vacuum tube caused the tube to glow. Crookes, in 1879, working with tubes of much higher vacuum (one-millionth of an atmosphere), discovered that luminosity was produced at the negative end of the tube; and in 1895 Röntgen, finding that the rays from a Crookes' tube caused barium cyanide to fluoresce, was led to further experiment which culminated in the discovery of Röntgen rays. The nature of the connection between Crookes' cathodal rays and Röntgen rays was settled by J. J. Thomson, who proved that the former consisted of a stream of negatively charged material particles, the impact of which against the glass gave rise to a series of disconnected pulses without periodicity—the X-rays. The practical applications of Röntgen's discovery were so evident as at once to stimulate fresh research, and in 1896 Becquerel found that certain uranium compounds which have the property of fluorescence affected a photographic plate in the dark, even if they had ceased to fluoresce through not having been exposed to sunlight for some days. Following up this clue, Mme. Curie found that pitchblende, whence uranium is obtained, was even more radio-active than the metal itself. After a laborious research she showed that this radio-activity was due to two new elements—polonium, which about equals uranium, and *radium*, which in its pure state is about two million times as active.

Radium belongs to the alkaline group of metals, and is intermediate between barium and thorium; it has not been isolated in the pure state, but if it was it would probably behave like sodium and oxidise rapidly. The bromide and chloride of radium are the salts employed. These are extracted from pitchblende refuse (uraninite) from which the uranium has been removed. The final separ-

ation of the radium from the barium and calcium chloride with which it is mixed is effected by a process of fractional crystallisation. In the successive stages of the isolation of the substance Mme. Curie made use of a suitable electroscope by which the ionisation of air by radio-active bodies could be detected. In this way she learned whether the radio-active substance she sought was in the precipitate or in the filtrate after each chemical operation. Pure radium chloride is a white crystalline body with an atomic weight of 225. The amount present in different minerals bears a constant relation of 1 : 1,000,000 to the uranium which they contain. A ton of Joachimsthal uraninite should yield about ·17 gramme of radium.

Here only the more important properties of radium can be mentioned. The whole question of radio-activity is extremely complicated, and for authoritative information concerning the latest theories it involves, reference should be made to Rutherford's *Radio-active Transformations* (London, 1906). Radium has the peculiarity of maintaining itself at a temperature of 2°-3° F. above the surrounding air; in other words, it gives off enough heat every hour to melt its own weight of ice, and goes on doing this indefinitely, without undergoing any appreciable change. It has the further power of ionising gases—that is, it converts the air between the terminals of an electric current into a conductor; this property is made use of in measuring radio-activity. It has, as a third peculiarity, the power of rendering certain fluorescent bodies luminous. It also induces temporary radio-activity in substances placed near it. This is due to an emanation of radio-active gaseous material which is deposited as a non-gaseous type of matter on all bodies in the neighbourhood. Thus the processes taking place are very complicated. "In a compound of radium there occurs a rapid expulsion of α and β particles, accompanied by the generation of γ rays, a rapid emission of heat, the continuous production of an emanation or gas, and the formation of an active deposit which gives rise to 'excited' activity" (Rutherford). The most feasible explanation of these phenomena is afforded by the "disintegration theory" of Rutherford and Soddy. "It is supposed that the atoms of the radio-active bodies are unstable, and that a certain fixed proportion of them become unstable every second and break up with explosive violence, accompanied in general by the expulsion of an α or β particle or both together. The residue of the atom, in consequence of the loss of an α particle, is lighter than before, and becomes the atom of a new substance quite distinct in chemical and physical properties from its parent."

The radiations are divided into α, β, and γ radiations, which agree in acting upon photographic plates, in exciting phosphorescence, and in discharging electrified bodies; they disagree in many important respects. The α rays consist of positively charged material particles about twice the size of a hydrogen atom projected at the rate of 20,000 miles a second; they do not penetrate a plate of aluminium ·05 mm. thick, and are slightly deflected in a magnetic field. The β rays also consist of material particles; they are only $\frac{1}{1000}$ of the size of the α particles, are negatively charged with electricity, and are projected at the rate of 1,000,000 miles a second. They penetrate 5 mm. of aluminium, and are readily deflected in the magnetic field. The γ rays are not material; their velocity is enormous, they can penetrate 50 cm. of aluminium, and are not deflected in the magnetic field. Rutherford and Soddy have shown that the α particles, if not identical with atoms of helium, are at least very closely related to that element. The β rays are identical with the cathodal stream, and the γ rays nearly resemble the penetrating X-rays produced by a high vacuum tube. They are thus a type of wave-motion in the ether dependent on the expulsion of the β particles.

Bromide and chloride of radium emit very little emanation in a dry atmosphere. The emanation is stored up in the mass of the compound, but is released by heat or solution. Radium which has thus been freed from emanation suffers a diminution in its activity as measured by the α rays, but this loss is recovered from as more emanation is produced and stored up in the mass. The emanation itself behaves like a heavy gas, but is not permanent and undergoes disintegration with the further expulsion of α particles and their deposit on surrounding bodies. The emanation is about 100,000 times as active as radium, and its transformation is accompanied by an enormous evolution of heat. It is "the concentrated essence of the radio-activity of radium." A "tube containing the radium emanation . . . emits α, β, and γ rays, evolves heat, and produces luminosity in many substances. Radium itself, freed from the emanation and the active deposit, emits only α rays" (Rutherford).

Medical Uses of Radium.—On the whole, the medical uses of radium are not very dissimilar to those of the X-rays. Owing to its cost, and the small amount in which it is procurable, it is not practicable to employ it as a means of taking skiagrams, although experimentally the procedure is feasible enough. The same circumstances also militate against its general use in other ways.

Physiological Effects.—Exposure to the radiations causes dermatitis, which may go on to intractable ulceration. A latent period of 8 or 10 days elapses before these effects are produced. Exposure to radium of 1,000,000 strength for 3 hours will cause dermatitis; for 10 hours, ulceration. When radium is brought

near the closed eye a sensation of light is produced. This is also perceptible in some cases of blindness. It is not, however, due to stimulation of the retina or optic nerve, but to fluorescence of the ocular media. Radium is inimical to animal life. The introduction of a tube containing 1 mg. of the bromide under the skin of a mouse causes convulsions, paralysis, and death. Young mice lose their hair, become blind, and die under its influence. After prolonged exposure, seeds lose their power of germinating; the growth of tadpoles is checked; other larval forms of life also fail to develop. The radiations are said to inhibit the growth of some bacteria and to kill others. Attempts to sterilise milk in this way have failed. It should be noted that the action is greatest in young forms of life—a fact which may be related to the rather selective effect of the rays on cancer cells. Thies found that the rays affect all animal tissues, the adenoid structures and skin being most susceptible. The epidermis dies, or under certain circumstances undergoes proliferation and produces a species of neoplasm. The arteries contract; striped muscle undergoes colloid degeneration; elastic fibres are most resistant. Heinecke, to whose work so much of our knowledge of the tissue changes caused by X-rays is due, states that the effects of radium are identical: the action on the hair, skin, or testicles is the same in both cases.

Therapeutic Uses. Method of Application.—Tracy advises the use of the most active preparation obtainable—at least 10 mg. of pure radium bromide of about 1,000,000 strength. Weaker samples necessitate longer exposures. Using pure radium bromide, the average sitting is from 20 to 45 minutes. Other authorities advise much shorter exposures—1 to 4 minutes. The radium may be enclosed in a glass or aluminium capsule, the latter being more permeable to α and β rays. A lead capsule with an aluminium window may also be used. The radium is placed in contact with small lesions; at a short distance from larger ones. Exposures may be made daily for one or two weeks, then thrice weekly. Malignant disease and lupus require long exposures often repeated; nervous affections fewer and shorter.

There is no doubt that, as Butcher graphically expresses it, small superficial tumours of all kinds "wilt and wither away" after a few exposures. The action is like that of X-rays, there being first spasm of the vessels, then congestion, cutaneous reaction, and sometimes pigmentation. In advanced malignant disease the ulceration may be controlled and fœtor diminished. It would seem therefore that radium may be tried with some prospect of success in the same class of case as X-rays. Good results have been reported in superficial epitheliomata, rodent ulcer, lupus vulgaris, lupus erythematosus, scleroma of the nose and larynx, angiomata, keloid, melanoma, by Lassar and many others. It has also advocates in the treatment of tics, neuralgia, and optic atrophy. Its advantage over X-rays lies in the possibility of applying it to localities which are impossible or difficult of access for these. Walker praises it highly in lupus of the mouth; it has also been employed in tuberculous ulcers of the tongue and in cancer of the œsophagus, with relief. Dinger reports cases of trachoma successfully handled with radium. Some remarkable observations of Tizzoni and Bongiovanni demand mention. They find that the virus of rabies can be rendered inert by exposure *in vitro* to the radiations, and that even after inoculation into a rabbit it can be rendered harmless in the same way. It is even stated that when the inoculation is made at a distant part — the meninges or sciatic nerve—raying one of the animal's eyes may neutralise the toxic action; and also that by exposure to the rays the virus is converted into a protective vaccine.

Other Forms of Radio-activity.—The property of radio-activity is possessed by other bodies besides radium, and appears to be very widely distributed. It has been conclusively proved that the water of many mineral springs —*e.g.* Bath—is radio-active, and the suggestion has been made that part of their virtues may arise from this. Taking advantage of the "excited activity" which radium induces, a radium fluid, consisting of normal saline solution which has been exposed to the action of the rays, and has thereby acquired radio-active properties, has been used for douches, gargles, and other purposes. Of a number of radio-active bodies, the only ones which need be mentioned are uranium and thorium. Both are far inferior in activity to radium—uranium, indeed, gives the unit standard of activity—hence no danger can follow their use. Uranium has been employed as a plaster in skin diseases (Walker). Thorium, which is the principal ingredient in incandescent gas mantles, was recommended by Rutherford and Soddy as deserving trial in pulmonary tuberculosis. The emanation is inhaled from a special apparatus (Lieber) in which thorium oxide is heated over a sand-bath. Thorium has also been employed as an ointment in malignant and other forms of ulceration.

Promising as the radio-active bodies may appear in the cure of disease, they are still on trial. It should never on any account be forgotten that, so far as malignant disease is concerned, the appeal is still to the knife. The sphere of radio-activity is only to supplement, never to supplant, early and thorough removal of the disease by operation.

Radius. *See* Elbow-Joint, Injuries and Diseases of (*Fracture of the Head or Neck of the Radius, Dislocation of the Radius*, etc.); Frac-

TURES (*Radius and Ulna*); WRIST-JOINT INJURIES (*Fractures*, etc.).

Radix.—A root (Lat. *radix*, a root), origin, or source. Thus the root of the nose is called *radix nasi*; and in Materia Medica the word often occurs, as *radix belladonnæ*, *radix rhei*, etc.

Rag-Picker's or Rag-Sorter's Disease. *See* ANTHRAX.

Ragatz-Pfaefers. *See* BALNEOLOGY (*Switzerland*); MINERAL WATERS (*Thermal Waters*).

Railway Brain. *See* RAILWAY SPINE.

Railway Sickness.—The nausea or actual sickness produced in sensitive persons by the oscillation of the carriage, etc., in railway travelling.

Railway Spine. — Concussion of the spine, especially that produced by a railway accident. *See* SPINE, SURGICAL AFFECTIONS OF (*Railway Spine*).

Rain Gauge. *See* METEOROLOGY (*Measurement of Rainfall*).

Rainey's Bodies, Corpuscles, or Tubes.—Psorosperms, found specially in the muscles of the pig. *See* PARASITES (*Protozoa, Sarcocystis*).

Rainfall. *See* CLIMATE (*Individual Factors*); DIPHTHERIA (*Etiology, Climatic Conditions*); METEOROLOGY (*Rain and Snow, Rainfall*).

Râle.—A rattling sound (Fr. *râle*, a rattle) produced in the lungs and heard on auscultation; it is due to the passage of air over or through mucus in the respiratory passages (larynx, trachea, and bronchi); different varieties are described, under the names of bubbling, coarse, crepitant, dry, fine, moist, sibilant, and sonorous. *See* CHEST, CLINICAL INVESTIGATION OF THE (*Auscultation of the Lungs*).

Rami. — Branches (plural of *ramus*, a branch); *e.g.* the *rami communicantes* of the spinal nerves, the *ramus* of the ischium, of the lower jaw, or of the pubes.

Ramollissement.—Softening of a tissue or organ occurring as a morbid process.

Randamel. *See* BALNEOLOGY (*Norway, Sweden, and Iceland*).

Ransom's Disinfector.—An apparatus for disinfecting by means of hot dry air.

Ranula.—A small cyst situated below the tongue, and due to occlusion of a gland duct; it may be congenital. *See* MOUTH, DISEASES OF (*Diseases of the Floor of the Mouth, Ranulas*); PAROTID GLAND, DISORDERS OF (*Inflammatory Conditions, "Acute Ranula"*); SALIVARY GLANDS, DISORDERS OF (*Ranula*).

Ranvier's Nodes. *See* PHYSIOLOGY, TISSUES (*Nerve, Structure*, Fig. 38).

Ranvier's Solution. — A saturated solution of iodine containing 2 per cent of potassium iodide; it is used for examining red blood corpuscles in a dry clot, for it does not lake them or alter their shape.

Rapallo. *See* THERAPEUTICS, HEALTH RESORTS (*Riviera*).

Rape. *See* MEDICINE, FORENSIC (*Rape*).

Raphe.—A ridge or seam (Gr. ῥαφή, a seam), such as the middle line of the scrotum, tongue, and palate.

Rapport. *See* HYPNOTISM (*Experimental Phenomena, Rapport*).

Rasch's Sign.—Uterine fluctuation, or the cyst-like sensation in the uterus detected during pregnancy by the bimanual examination.

Rashes. — Eruptions, cutaneous or on mucous membranes, as seen in fevers, purpura, poisoning, etc. *See* DIPHTHERIA (*Antitoxin Treatment, After Effects*); DRUG ERUPTIONS; GLANDERS (*Symptoms*); MORPHINOMANIA AND ALLIED DRUG HABITS (*Antipyrin Rashes*); OVARIES, DISEASES OF (*Rashes after Ovariotomy*); PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Cutaneous Rashes*); TYPHOID FEVER (*General Symptoms*); TYPHUS FEVER (*Period of Advance*).

Raspberry Tongue. *See* SCARLET FEVER (*Symptomatology, Tongue*).

Rathke's Gland.—Jacobson's organ in the nasal septum.

Rathke's Pouch.—A diverticulum in the pharynx of the embryo connected with the anterior part of the pituitary body; the bursa pharyngea (of the adult) perhaps represents its remains.

Rats. *See* ANTHRAX (*in the Lower Animals*); PLAGUE (*Dissemination by Rats*).

Rat-tail Tendon. *See* ASEPTIC TREATMENT OF WOUNDS (*Sutures*).

Ratzes. *See* BALNEOLOGY (*Austria*).

Rauber's Layer.—The flat cells of the enveloping layer which cover the inner cell mass of the ovum during segmentation.

Raw Meat. *See* SCURVY, INFANTILE (*Treatment*); INFANT FEEDING (*Meat Foods, Raw Meat Juice*).

Ray Fungus. *See* Actinomycosis.

Raynaud's Disease.

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Erythromelalgia

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See also Gangrene (*Varieties, Symmetrical Gangrene*); Gout (*Irregular, Manifestations in Nervous System*); Hæmoglobinuria (*Paroxysmal*); Nails, Affections of the (*in Raynaud's Disease*).

Although instances of this disease have been recorded in medical literature as far back as Schrader's case in 1679, it was not until the year 1862 that the French physician, Maurice Raynaud, called attention to the group of symptoms which has since borne his name. Like migraine, epilepsy, and the so-called "functional" diseases, Raynaud's disease is a clinical, not a pathological entity. Whilst it forms a striking clinical picture, and is doubtless dependent on some faulty innervation of the vascular system, yet no constant structural changes have been found, either in the nervous or vascular tissues, which can adequately explain the profound morbid appearances presented by this affection.

Etiology.—*Sex.*—The disease is commoner in the female sex, the proportion (both in Munro's 180, and in Cassirer's 104 collected cases) being 1·7 females for every male. The *age* of onset, in the vast majority of cases, is under thirty years, the average of 171 cases being 28·9 years (Munro). From the striking frequency with which young adults are affected, Raynaud was inclined to apply the term "juvenile gangrene" to the affection. But no age is exempt. Infancy and old age alike have been attacked, the youngest recorded case being that of an infant aged six months (Friedel), the oldest that of a woman of seventy-seven years (Henry). A *neuropathic diathesis* seems to be the most important predisposing factor. Raynaud himself pointed out that nervous individuals are chiefly affected. Direct heredity, on the other hand, where parent and child have both suffered from the disease, is uncommon. The most frequent exciting cause is *exposure to cold and damp*, and such patients are often abnormally sensitive to cold. Thus, as might be expected, the complaint is commoner in winter than in summer, and occurs more frequently in people whose occupations entail exposure to cold and damp, *e.g.* washerwomen. Many cases, however, occur in which no such exciting cause can be found. Fatigue, especially when combined with exposure, may bring on an attack; so also may *mental shock or emotion*. There is no evidence that disorder of the sexual organs has any causal relation to the complaint. Raynaud's disease may also supervene during the course of other chronic affections such as malaria, congenital syphilis, gout, chlorosis, diabetes, and paroxysmal hæmoglobinuria. A few cases have been recorded in which a *toxic* influence appears to have been at work, as when the symptoms have developed for the first time during an attack of intestinal obstruction.

Symptoms.—*Summary.*—The chief symptoms are pains, more or less severe, and paræsthesia of various sorts. They are not confined to the area of distribution of definite nerve trunks, but affect, usually, the periphery of the limbs (more commonly the upper extremities). Less commonly they occur in other situations, such as the ears, lips, nose, etc. The distribution is generally symmetrical. To these symptoms are superadded signs of paroxysmal vaso-motor disturbance in the form of (1) *local pallor* ("*syncope*"), or (2) *local cyanosis* ("*asphyxia*"), less commonly redness. After a time, or even in some cases without antecedent vaso-motor phenomena, trophic lesions occur in the affected parts, mostly of the nature of (3) *gangrene*. These three stages of the disease may occur either successively, or the first and second may occur without the third, and conversely. The course of the disease is an intermittent one. Anæsthesia and motor paralysis as a rule are absent. During the intervals between the paroxysms there may either be persistent signs of faulty vaso-motor innervation, or there may be no such signs or symptoms. The disease may exhaust itself in a single paroxysm, or many successive attacks may occur.

(1) *Local Syncope.*—In this form of Raynaud's disease the affected parts, usually the fingers (less frequently the toes, sometimes the margins of the pinnæ, the tip of the nose, or in rare cases even the tip of the tongue) suddenly become cold to the touch and white in colour. The degree of pallor varies; it may be waxy, tallowy, or corpse-like; sometimes it is bluish-white. What usually attracts the patient's attention, however, is not so much the change of colour, as some sensory disturbance in the form of pain or paræsthesia, *e.g.* numbness, tingling, feeling of stiffness, or an indescribable consciousness that something unusual is going on in the part. As the pallor increases, so does the pain or paræsthesia become more intense, and

at this stage objective blunting of sensation to touch can often be recognised. Marked anæsthesia to touch may coexist with severe subjective pain. The power of movement of the affected fingers or toes may be temporarily impaired. This is probably not a true motor palsy, but due to deficiency of afferent impressions causing clumsiness of the fingers. In exceptional cases slight secretory phenomena have been observed in the form of cold perspiration in the affected parts.

The distribution of these phenomena varies. It may be unilateral or bilateral. Sometimes it is so exactly symmetrical that the identical digits are affected in both hands. The terminal phalanx alone may be attacked, or the condition may extend upwards along two or more phalanges, or may even, in rare cases, involve the whole hand or foot. The thumb frequently escapes, the other fingers seeming to be equally liable. One or more fingers may be affected at a time.

The duration of a paroxysm may be momentary or may last for hours. Numerous attacks —twenty to thirty per diem—may occur in rapid succession, or the paroxysms come at longer intervals:—weekly, monthly, or perhaps only in winter. The return to normal after an attack also varies in different cases. In some it is sudden, in others pallor is followed by a stage of reaction with tingling, burning pains, and local heat and redness. Or the condition may pass on to the second stage—that of local cyanosis.

In its mildest form local syncope is popularly known as "dead finger," a condition not uncommon in nervous people and in otherwise healthy individuals on exposure to cold, mental excitement, etc.

In predisposed individuals the attack often comes on whilst washing the hands in the mornings, or while performing fine digital movements, such as piano-playing or sewing.

As a rule, during an attack the accessible arteries of the limb are not perceptibly altered in calibre, though occasionally the pulse may be reduced in volume. In a few cases hæmoglobin has been found free in the blood-plasma of the affected finger (local hæmoglobinæmia).

Repeated attacks tend to make the fingers thin, parchment-like, and conical—sclerodermia. In spite of numerous attacks, however, the general health is not necessarily impaired, and the condition remains a purely local one.

(2) *Local Asphyxia or Cyanosis.*—This is a stage of the disease in which there is sudden discoloration of the skin, varying in tint from a dusky blue or slate-colour to an intense purplish-black. Sometimes an admixture of red may be present. Pressure on the discoloured part causes a white mark which persists for some time, lividity returning to it with great slowness. The parts adjacent to the affected area often show a bluish mottling, which in exceptional cases spreads over the whole body.

The local temperature falls during the paroxysm to an extent corresponding to the duration of the cyanosis.

A slight degree of swelling, not œdematous nor indentible on pressure, is fairly common. If the cyanotic area be pricked, dark blood escapes, unless gangrene be about to set in, when no blood flows. The veins of the cyanotic parts are sometimes distended; in rare cases they have been seen spasmodically contracted, and Sir T. Barlow has observed a beaded appearance, rapidly shifting in position, in the veins. In a considerable number of instances the cyanosed part sweats freely, especially when it is beginning to regain its normal colour.

Local cyanosis may either develop directly from the normal, or it may follow an antecedent stage of local syncope. Pallor and cyanosis may succeed each other, and conversely, in the same patient. Or there may be pallor of the distal phalanx with cyanosis of the proximal, or *vice versa.*

As the paroxysm of cyanosis subsides—which it may do after a period varying from a few minutes to several hours—there may either be an interval of active hyperæmia or a gradual subsidence of the lividity by the appearance in the dusky area of bright spots, gradually coalescing. Other cases, again, pass directly from the stage of cyanosis into that of gangrene.

Cases of sclerodermia from Raynaud's disease nearly always manifest at some stage the symptoms of local asphyxia.

Subjective symptoms are sometimes absent; more often, however, during a paroxysm there is pain of a burning or stinging description, which may be of agonising intensity.

Anæsthesia, analgesia, or thermanæsthesia may be present, separately or conjointly, or hyperalgesia may occur. On the other hand sensation is sometimes unaffected.

As to distribution, whilst local syncope seldom occurs except in the limbs, local cyanosis, though preferring the limbs (and the upper rather than the lower), frequently attacks the ears and face. The distal parts of the limbs, ears, and nose are most commonly affected; the cheeks, chin, and tongue less frequently. Patches of lividity on the trunk or on the proximal segment of a limb are uncommon. The order in which different parts are attacked is extremely variable, and is not always the same in successive paroxysms. As a rule the parts affected are roughly symmetrical, though unilateral paroxysms also occur.

(3) *Symmetrical Gangrene.*—In this variety of the disease trophic changes are superadded to vaso-motor phenomena, gangrene being the final stage towards which the others tend. It is usually preceded by local asphyxia, and often by local syncope also.

The parts most frequently involved are the extremities of the limbs (especially the upper limbs) and of the ears, unilaterally or bilaterally. The thumbs are less liable than the other digits. In exceptional cases gangrene attacks other parts, such as the cheeks or chin, and in rare instances the penis. Labia majora, nipples, lips, eyelids, tissues over the coccyx, or the proximal parts of the extremities have also been affected. Instances have occurred where large areas of the limbs have been attacked. Thus in one of Barlow's cases amputation had to be performed in the middle third of the calf.

As a rule the gangrenous process is confined to a small part of the cyanosed area. In its mildest form the necrosis may be limited to the epithelium, so that only desquamation results. More often a small blister forms, with sanguineo-serous or dark contents. This bursts, causing an excoriation or ulcer in the skin which, on healing, leaves a scar behind. The nails may be lost and subsequently reproduced. If the necrosis extends deeper into the cutis and subcutaneous tissue, large blebs form which, as the subjacent tissues necrose, become deep brown. The slough ultimately separates and leaves a slowly healing ulcer, sometimes with sharply punched-out margins.

The soft tissues may necrose to any depth, and whole phalanges thus undergo dry mummification. A line of demarcation forms, and the gangrenous part is cast off, leaving a conical stump behind. As a rule the bones escape necrosis. The resulting scar often remains for weeks crusted with scabs, which tend to re-form on removal. Moist gangrene is rare.

Gangrene and cyanosis may be present simultaneously in the same limb, and the three stages of syncope, cyanosis, and gangrene may be observed at one time in different digits. This, however, is uncommon. The distribution is roughly symmetrical, though not necessarily equally intense on the two sides. There may be severe gangrene in the limb of one side, with an insignificant blister on the other.

Several types of cases occur: in one variety attacks of local asphyxia occur, in which the cyanosis becomes constant instead of intermittent, and in the dusky area a part necroses and is cast off. In another type paroxysms of local syncope or cyanosis occur, and small blisters, ulcers, or sloughs develop in the affected digits. In others the cyanosis is chronic from the outset, especially during the winter months, not paroxysmal, and small blebs or ulcers appear in the cyanotic area. In rare cases symmetrical gangrene has occurred without previous cyanosis.

Pain is usually a marked feature, especially during the preceding paroxysmal cyanosis. It is often of excruciating intensity, not confined to the gangrenous parts, but radiating from them up the limb. Yet in some cases cyanosis goes on to gangrene quite painlessly; this, however, is exceptional.

Progress and Complications.—Raynaud's disease usually occurs in a succession of paroxysms, the duration of each varying from a few minutes to many hours. A large number of cases never go on to gangrene, but consist merely of attacks of local syncope and local cyanosis, or of either of these alone. Some of the less acute cases, *e.g.* after acute fevers, recover permanently and completely. Some cases, even of gangrene, have only a single attack and do not recur.

Amongst the various *complications* which may occur, one of the most important is *sclerodermia* of the fingers, which may precede or succeed the Raynaud's disease, though it often occurs totally unassociated with the latter. Other diseases of the skin are also occasionally associated with Raynaud's disease, amongst which may be mentioned urticaria, cutaneous hæmorrhages, and angio-neurotic œdema. Joint effusions, with subsequent ankyloses, especially of the smaller joints, have been observed.

But complications of a less local character also occur. Thus sometimes one of the acute infective fevers has been an antecedent. Other cases give a history of *malaria*, sometimes many years before. Such cases would point to the *toxic* origin of some cases of Raynaud's disease.

Numerous urinary abnormalities have also occurred, of which the most important is *paroxysmal hæmoglobinuria*. This has been noted in over 6 per cent of cases. The connection between the two diseases is regarded by some as a very close one, so much so that they consider the two states as essentially identical; both being paroxysmal and both excited by cold. Others, however, regard hæmoglobinuria as a secondary complication, since it occurs not only in Raynaud's disease, but also in the course of other morbid conditions, such as malaria and syphilis. Malaria is certainly a fairly common antecedent of hæmoglobinuria. It is conceivable that in certain cases of local cyanosis a local hæmoglobinuria is produced (indeed, in a few cases free hæmoglobin has been demonstrated in the blood-serum obtained from the affected finger during a paroxysm), and that this causes the subsequent appearance of hæmoglobin in the urine. But in some cases hæmoglobinuria precedes the local cyanosis, or is concomitant with it, and if there be a cause common to the two symptoms it must be a central one.

Albuminuria, constant or intermittent, has been observed, the latter being rare and sometimes combined with hæmoglobinuria. Diabetes insipidus and glycosuria have been recorded in a few instances, but the possibility of diabetic gangrene should always be carefully borne in mind.

Cardio-vascular disease has been noted in fully 12 per cent of cases. Some of these are, no doubt, mere accidental coincidences. Epistaxis

sometimes occurs. Syphilis, hereditary or acquired, has been noted in a few cases, in certain of which antisyphilitic treatment relieved the vaso-motor symptoms.

Diseases of the nervous system are relatively common, being present in nearly one-fourth of reported cases. Hemiplegia is the commonest variety of nervous disease, but hydrocephalus, acromegaly, spinal tumour, and tabes dorsalis, or such complaints as hysteria, chorea, epilepsy, and vertigo, have occurred, and the disease is certainly most frequent in neuropathic individuals. Symptoms of peripheral neuritis are rare, and the pathological changes described in the peripheral nerves are probably the result and not the cause of the disease. Psychical affections, such as mania, melancholia, or dementia, are sometimes present. *Paroxysmal impairment of vision* has been recorded, sometimes alternating with the attacks of local cyanosis. During such visual impairment the retinal arteries have been seen ophthalmoscopically to be in a state of contraction.

Morbid Anatomy. — No constant morbid changes have been authenticated in the vascular system or in the central or peripheral nervous system, and although endarteritis, arterial thrombosis, endophlebitis, and neuritis have all been described, yet more often the vessels and nerves have been found normal. Cases in which pathological changes have been found have chiefly been in limbs amputated for gangrene, in which the morbid appearances were probably consequent to the gangrene and not its cause.

Pathology.—The paroxysmal nature of the vascular disturbances points, as Raynaud himself stated, to a central rather than a peripheral source for the disease.

Local syncope is undoubtedly due to a local spasm of the vessels, its sudden appearance and disappearance being incompatible with any other hypothesis.

The occurrence of local cyanosis has been variously explained. Raynaud attributed it to inequality between the spasm in the veins and that in the arteries, the veins regaining their normal calibre whilst the arteries still remain contracted, the cyanosis being due to the reflux of venous blood into the capillaries. He recalled the observation, in this connection, of contraction of the retinal arteries during certain cases of local cyanosis.

But local asphyxia often arises without a previous stage of local syncope. This can hardly be accounted for otherwise than on the hypothesis of a primary venous spasm, causing capillary stasis and cyanosis. Waves of venous spasm have actually been observed in certain cases. Hutchinson holds that the pathological process is not the same in every case, and that, just as in some healthy people cold causes pallor, whilst in others cyanosis or redness is produced, so in Raynaud's disease, if the spasm is chiefly arterial, pallor results—if chiefly venous, cyanosis occurs.

The sensory symptoms in the affected parts are probably due to imperfect blood-supply. The trophic changes, in the form of gangrene, are more difficult of explanation. Mere vascular spasm, however prolonged, does not readily produce necrosis of tissue, though it may seriously damage its vitality and render it less resistant to slight traumatisms. Those cases of gangrene preceded by cyanosis, but without local syncope, must be associated with an excess of venous blood, and yet profound cyanosis in other parts of the body is possible without producing gangrene. Probably the gangrene of Raynaud's disease is neuropathic in origin and analogous to the glossy skin of nerve injuries or to the bed-sores of acute myelitis.

The symmetry of the affection and its paroxysmal nature clearly point, as Raynaud indicated, to a central affection, probably an increased excitability of the vaso-motor centres, rather than to any mere peripheral lesion. The disease is thus an *angio-neurosis*, in which the nervous system is primarily at fault.

The cause of such increased excitability of the vaso-motor centres appears to vary in different cases. Thus the condition may be congenital in cases where there is a nervous heredity, or it may result from various pathological influences, for example cold, long-continued or frequently repeated. Or it may be the direct result of toxins acting on the nerve centres, as in fevers, syphilis, malaria, or intestinal obstruction, or it may conceivably be reflex from peripheral sources. Finally, Raynaud's disease may occur in the course of organic diseases of the central nervous system, in which, along with other structures, the vaso-motor centres happen to be implicated.

Diagnosis. — A well-marked case of Raynaud's disease usually presents no difficulty in diagnosis; it is chiefly with abortive or atypical forms of the disease that one is in doubt. Thus a paroxysm of *erythromelalgia* may resemble the rarer variety of Raynaud's disease in which redness is present instead of cyanosis. In some cases the diagnosis between the two conditions is so difficult as to be impossible, and the greater the number of cases observed, the more arbitrary does the division between them appear. The occurrence of trophic changes, however, in a doubtful case would tend to place it under the category of Raynaud's disease.

Morvan's disease, a variety of syringomyelia, with marked trophic changes in the fingers and toes, is not paroxysmal, but of slow onset. It is less strikingly symmetrical than Raynaud's disease, and is, moreover, accompanied by dissociated sensory paralysis and often by muscular atrophy. Painless abscesses and whitlows, together with necrosis of bone, are common in

Morvan's disease, whilst in Raynaud's disease they are rare.

Frost-bite may at first sight be difficult to distinguish from Raynaud's disease, all the more so since the latter condition is often precipitated by cold. A symmetrical distribution, however, is against frost-bite, and a history of previous attacks at a time of year when there was no frost, and especially the paroxysmal nature of the symptoms, would indicate Raynaud's disease.

In chronic *ergotism* (an exceedingly rare affection) the condition is usually epidemic, and there is a history of contamination of rye bread.

Senile gangrene usually affects the whole foot, and is rarely symmetrical, or attacking individual toes, whilst Raynaud's disease quite as often affects the upper as the lower extremities, is more superficial as a rule, and generally affords a history of previous attacks.

Diabetic gangrene is generally of the moist variety, and is associated with endarteritis, neuritis, and glycosuria.

Leprosy, beri-beri, congenital cyanosis, and hysterical blue œdema need only to be mentioned to be at once distinguished from Raynaud's disease.

Prognosis.—Raynaud's disease is not of itself dangerous, nor does it shorten life. The paroxysms, however, tend to recur again and again, especially in cases of local cyanosis. Cases which go on to gangrene tend ultimately to pass off. The prognosis in individual cases is difficult, inasmuch as slight cases may afterwards go on to gangrene, whereas threatened gangrene may become arrested.

Treatment.—Little can be done beyond the avoidance of cold and keeping the extremities warm during the attack. The patient should wear warm woollen gloves, not too tight. During a paroxysm the pain may require morphia. Other drugs, such as atropin, chloral, or the nitrites, have proved of little avail. Raynaud recommended galvanism to the spine, and obtained encouraging results in certain cases. Barlow advocates local galvanism, the limb being immersed meanwhile in salt water, into which one electrode is led, the other being applied higher up the limb. Gentle massage between the paroxysms is to be recommended. Counter-irritant applications must be avoided. In malarious cases quinine should be exhibited, in cases with arterio-sclerosis iodide of potassium, and in syphilis anti-specific treatment should be carried out. Gangrene should be dealt with on surgical principles.

Erythromelalgia

The group of symptoms included under this name, originally described by Weir Mitchell, of Philadelphia, in 1872, consists essentially of paroxysmal pain, redness, and swelling, chiefly, though not exclusively, affecting the feet. Lewin and Benda, who collected 41 cases, were the first to point out, in 1894, that erythromelalgia is not a disease of itself, but occurs in the course of many nervous disorders. The affection is not a common one. Cassirer in 1901, after an exhaustive examination of the literature on the subject, had collected 90 instances in all.

Etiology.—*Sex.*—A slight majority of cases, as in Raynaud's disease, has occurred in males. The *age* of onset has mostly been in middle life, between the ages of twenty and sixty, though neither extreme of life is exempt. *Occupations* which entail severe exertion with long hours of standing, or exposure to varying temperatures, appear to act as remote predisposing causes. Thus tailors, who have to sit in an awkward position at work, and fishermen, engine-drivers, and washerwomen, who are exposed to cold and wet, have been specially frequently affected. Previous illnesses may predispose by lowering the general vitality, but the symptoms often appear in apparently healthy individuals. Toxic causes, such as syphilis, alcoholism, malaria, etc., do not play a part of much importance. Local traumatism has occasionally preceded an attack, whilst cessation of the menses, whether accidental, as from falling into a river, or physiological, as at the menopause, has also been noted as preceding the first attack.

Symptoms.—The chief symptom consists in a peculiar burning or stabbing *pain*, generally affecting one foot, sudden or gradual in onset, and often of excruciating intensity. This pain is intermittent, and recurs at varying intervals in attacks lasting from a few minutes to several hours at a time. It is made worse by a dependent posture of the limb, by voluntary movement, or by warmth, whilst it is relieved by a horizontal posture, by rest, and by cold applications.

Redness and *swelling* of the affected area of skin are superadded, though often not until several weeks or months after the first appearance of the pain. More rarely the redness and swelling precede the pain. The redness is of a bright tint. Its area may be diffuse; more often it is circumscribed at first, affecting, for example, the ball of the hallux or of one of the other toes, the heel, or the outer or inner side of the foot. Sometimes several discrete patches occur. This redness spreads, increases in intensity, and, if the vertical position of the limb be maintained, there appears turgescence of the skin with bounding arteries and venous dilatation. This swelling of the skin occasionally pits on pressure; more usually this is not so. After some minutes or hours the "vascular storm" partly subsides, the hyperæmia becomes passive instead of active, the arteries throb less violently, and the bright red gives place to a bluish, dusky, cyanosed tint which persists for some little time.

During the red phase the pain becomes progressively more intense, and there is exquisite tenderness to all varieties of stimuli in the

affected part. The plantar reflex, in cases affecting the foot, is exaggerated or normal. The patient during a paroxysm takes off his shoes and stockings, or plunges the foot into cold water, or if he be in bed, finds even the pressure of the bedclothes on the foot intolerable.

The local temperature during an attack usually rises, and may be 2° or 3° C. higher than in the unaffected foot. Instead of the normal fall of local temperature on letting a limb hang down, the converse condition occurs, in which the temperature rises when the limb is dependent, and falls when raised to the horizontal posture. When cyanosis supervenes the temperature of the affected foot falls.

The above symptoms are paroxysmal, not continuous. The effect of the dependent posture in producing pain, redness, and swelling is often instantaneous, so that the sufferer refuses to walk, or, if he walks, treads only on the unaffected part of the foot, or may even, during a severe attack, go on his hands and knees.

The *distribution* of the symptoms is primarily asymmetrical. The feet are chiefly affected, the hands less frequently. The pain may spread up the limb as far as the knee or forearm. When more limbs than one are affected, all are not attacked at once, but first one and then another. When the hands suffer, the characteristic attitude is one in which they are crossed over the breast or raised above the head. Less frequent sites of the affection are the middle of the back, the area of the neck behind the mastoid process, the testicle, or the mucous membrane of the mouth and pharynx.

Besides the cardinal symptoms above described, various concomitant local symptoms may be present. Thus *secretory* changes in the form of excessive sweating of the affected area may occur during a paroxysm. *Trophic* changes in the skin occur in a certain proportion of chronic cases; thus the skin and subcutaneous tissues may become either thickened or atrophic and glossy, and the nails may become coarse in texture. In one case observed by the writer desquamation of the affected area occurred after each paroxysm.

As in Raynaud's disease, the motor functions remain unaffected. Slight diffuse wasting of the muscles may occur, but not in the area of individual nerves. Electrical reactions of degeneration do not occur, nor are adjacent nerve-trunks tender as in peripheral neuritis.

Atypical cases are sometimes seen in which the vaso-motor symptoms are intermediate between erythromelalgia and Raynaud's disease. Thus the pain of erythromelalgia may be accompanied by the pallor and subnormal temperature of "local syncope," or even by cyanosis. Or redness and heat may be preceded by pallor and coldness. Or the symptoms of erythromelalgia and of Raynaud's disease may follow each other in the same person.

Morbid Anatomy and Pathology.—Very few cases have afforded opportunities of pathological investigation, and in most of them observers have been content with the examination of portions of the nerve-trunks adjacent to the affected parts. Out of six such cases, in three the nerves were normal, one showed neuritis, and in another there was some degeneration in certain posterior roots of the cauda equina. In three of the six cases arteriosclerosis was present.

As to the pathology of the affection, although its most striking signs are doubtless vaso-motor in origin, yet the superaddition of sensory, secretory, and trophic changes indicates that the vaso-motor system cannot alone be to blame. The cause of the paroxysms must accordingly be referred to some irritative condition of various nervous centres. The site and nature of such irritation are still matters of speculation. They cannot be referred, as some have supposed, to the sympathetic alone, since the pain is entirely disproportionate to that which occurs in any lesion of the sympathetic chain. One must rather look for the site of the disease in some place where sensory and vaso-motor fibres are closely associated, that is, in the spinal cord or medulla. In support of such a view may be urged not only the paroxysmal nature of the affection, but also the fact that in not a few cases erythromelalgia is one of the earliest symptoms in organic diseases of the cord. The presence of erythromelalgia should lead the observer to ascribe any subsequent nervous phenomena to organic rather than to functional disorder.

It is not improbable that the site of the mischief is not always the same, and that in different cases the peripheral nerves, the spinal cord, or the medulla may be at fault. Thus those cases which follow more or less closely the distribution of peripheral nerves may be due to neuritis affecting especially the vaso-dilator and secretory fibres, whilst cases which are diffusely distributed over a whole limb or several limbs may be referable to some central cause, spinal or bulbar.

Diagnosis.—In well-marked cases this is usually easy. Painless vaso-motor changes should not be classed as erythromelalgia, and, conversely, pain alone is not diagnostic. As already explained, cases occur in which the demarcation from Raynaud's disease cannot be sharply drawn.

From neuritis it is distinguished by the absence of muscular atrophy and of anæsthesia, from ordinary neuralgia by the absence of the "tender points" and by the presence of the vaso-motor phenomena. Intermittent limp—*intermittirende Hinken*—is a condition associated with arteritis of the peripheral vessels of the

lower limbs, in which pains and paræsthesia come on in the legs on walking and pass off with rest. It is distinguished by the feebleness or absence of pulsation in the accessible arteries of the foot, and by the absence of the redness of erythromelalgia.

Prognosis and Treatment. — The disease, though chronic, is not of itself fatal. It may reach a certain degree of intensity and then become stationary; more often it is gradually progressive in intensity and distribution.

The treatment during a paroxysm consists in elevating the limb to a horizontal posture, the avoidance of movement, and the local application of cold.

Faradism in a few cases has been beneficial. In one case observed by the writer, a galvanic current, with the positive pole applied to the painful area, promptly relieved the pain.

Drugs are of minor importance, though phenazone, acetanilide, iodide of potassium, etc., have all been suggested. Morphia is a *dernier ressort*, and not rashly to be recommended. Operations on the peripheral nerves have been unsatisfactory, producing at the best only temporary relief, and in one case having been followed by gangrene.

Rays. *See* Beta Rays; Blondlot Rays; Radium; X-Rays; etc.

Ray's Mania.—General moral insanity, in which morbid changes in the cortex cerebri are found.

Reaction, Bordet's.—The biological test of blood, which may be thus defined: "The fact that the blood-serum of an animal into which has been injected the blood of another animal of different species develops the property of agglutinating and dissolving erythrocytes similar to those injected, but has no such effect upon blood derived from another source" (Da Costa).

Reaction, Löwenthal's.—A blood test for detecting relapsing fever in the stage preceding the appearance of spirilla in the blood; it consists in the mixing of the blood of a suspected case with that of a case in which spirilla are present and motile, when, if the suspected case be one of relapsing fever, the spirilla will become motionless and collect in masses.

Reaction of Degeneration. *See* Physiology, Tissues (*Muscle, Electrotonus*); Electricity (*Galvanism, Reaction of Degeneration*).

Reaction, Pfeiffer's.—The fact that the vibrios of Asiatic cholera when injected into an immunised animal lose their motility, break up, and dissolve; Pfeiffer's reaction was the foundation of Widal's test.

Reaction, Widal's. *See* Typhoid Fever (*Pathology, Serum Diagnosis*).

Reading. *See* Blindness (*Education of the Blind*).

Ready Method. — Marshall Hall's method of artificial respiration. *See* Asphyxia (*Resuscitation*).

Récamier's Curette. *See* Curettage (*History*).

Receptaculum.—A receptacle, such as the *receptaculum chyli* (dilated commencement of the thoracic duct), etc. *See* Physiology, Food and Digestion (*Absorption of Food, Channels*).

Receptors. *See* Physiology, Neuro-Muscular Mechanism (*Neural Arcs, Receptor Mechanisms*). *See also* Immunity, Recent Views on (*Ehrlich's Side-Chain Theory*).

Recessus.—A recess or depression, such as the recessus duodena-jejunalis, the recessus epitympanicus, the recessus pharyngeus, etc.

Recklinghausen's Disease. — The association of multiple fibromata, neuro-fibromata, and pigmentation of the skin. *See* Tumours of the Skin (*Melanotic Sarcoma*).

Reck's Disinfector.—A method of disinfection by the use of steam.

Reclination. *See* Cataract (*Treatment, Operations*).

Reclus's Disease.—Cystic disease or chronic cystic inflammation of the breast. *See* Mammary Gland, Diseases of (*Chronic Mastitis*).

Recognition. *See* Memory in Health and Disease (*Recognition*).

Recollection. *See* Physiology, Cerebrum (*Physiology of Memory*).

Rectal.—Relating to the rectum. *See* Rectum and *Cross References.*

Rectification. — Straightening a part, *e.g.* a deformed limb, or altering a presentation in obstetrics, *e.g.* a face case. *See* Labour, Management of (*Face Presentations*).

Rectified Spirit. *See* Alcohol (*Spirits*).

Rectitis. — Inflammation of the rectum; proctitis. *See* Dysentery (*Diagnosis*); Enemata (*Gangrenous Rectitis*); Rectum, Diseases of.

Recto-.—In compound words *recto-* (Lat. *rectus*, straight) means relating to the rectum; *e.g. recto-abdominal examination* (palpation of the pelvic organs by means of a finger in the rectum and a hand over the abdomen), *recto-colitis* (inflammation of the rectum and colon), *recto-cystotomy* (incision of the bladder carried out through the rectum), *rectoscope* (a rectal speculum), and *recto-vesical* (relating to the rectum and the bladder).

Rectocele.—Prolapse of the rectum into the vagina, *e.g.* in cases of procidentia uteri. *See* Pelvis, Perineum and Pelvic Floor (*Lesions of the Fixed Portion of the Pelvic Floor*); Uterus, Displacements of (*Descent*).

Recto-Vaginal.—Affecting the rectum and the vagina; *e.g.* a recto-vaginal fistula, the recto-vaginal septum, etc.

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Rectal diseases are among the most common that affect civilised humanity. Improper food and alcohol, sedentary indoor occupations, and defects in clothing have much influence in the causation of these maladies, which, though not usually dangerous to life, certainly give rise to a vast amount of suffering, and also to the distress arising from inability to work for daily bread.

The majority of these affections are very amenable to proper treatment. Some forms of rectal disease are much more common than others, notably fistula and piles.

In hospital practice fistula is more common than piles, but in private practice hæmorrhoids of various kinds are more frequently met with.

There are certain questions which it is desirable to ask the patient when investigating a case of rectal disease.

These refer to:—

(1) The presence or absence of pain, and its nature and position.

(2) Protrusion on action of the bowels, or on exertion.

(3) Nature of discharge if present.

(4) Constipation or diarrhœa.

It is not uncommon for stone, cystitis, prostatitis, or urethral stricture to give rise to rectal symptoms, which may be most prominent, and mislead a patient to suspect the rectum when the urinary organs are really the seat of his disease. In women the condition of the uterus must be ascertained.

Verbal interrogations concluded, an examination is to be made. The most comfortable and delicate position for the patient, and the most convenient for the surgeon, is obtained when the former lies on the right side, face and chest turned towards the couch, right arm behind the back, and knees drawn up to the abdomen. The buttocks are then in an oblique position, and leave the surgeon a good view of the anus. In special examination for growth or stricture the patient should be directed to stand up and bear down. Great gentleness must always be used in the examination. Note any discoloration and the condition of anus, whether patulous, contracted, or nipple-shaped. Look for tumour, ulceration, or fistulous openings; feel around the anus for induration. Press on the very verge of the anus, for a painful spot may be found indicating the position of a fissure. With the hands placed upon the buttocks, and the fingers near the anus, separate the former, telling the patient to bear down. The anus is thus everted, and the orifice and mucous membrane for half an inch up the bowel exposed to view. Fissure,

piles, or polypi are thus discovered. Now examine the interior of the bowel with the finger well anointed, the patient bearing down as it is inserted. The tightness and breadth of the sphincters are observed, and careful examination made in the space between them, the commonest position for openings of fistulæ. Then the finger is passed higher, and the prostate and upper parts of the rectum thoroughly explored.

If a tumour is felt, try to indent it with the finger. Scratch with the nail, to detect what matter is got from it; impacted fæces may give rise to symptoms of cancer, and blood, mucus, or pus on the nail helps diagnosis. Use the right forefinger for the front wall, and the left for the posterior aspect of the gut.

If the rectum be full of fæces obscuring diagnosis an enema is necessary. A speculum is seldom needed for diagnosis. When used it must be large, and an anæsthetic is required. Should nothing be found to account for the patient's symptoms, the highest part of the rectum and lower part of the sigmoid flexure may be examined by bougies with or without an anæsthetic. First india-rubber, then metal bougies are to be used. Passage of the hand into the rectum for diagnosis of disease high up is of doubtful value. In obscure cases it is most important to give an anæsthetic, dilate the sphincters, and examine the abdomen, pressing deeply into the left iliac fossa.

Abscess and Fistula in Ano.—Men are more subject to fistula than women. The disease is commonest in middle-age, but infants in arms, and patients over eighty, have alike needed operation for fistula. The commonest causes are:—

(1) Injury to the anus, as by passage of hard motions or foreign bodies.

(2) Exposure to wet or cold, as by sitting on damp seats after exercise, when the parts are hot and perspiring.

(3) Parasites of the anus and its neighbourhood. Thrombosed veins and suppurating piles are predisposing causes.

(4) Abscess and fistula may follow fevers, and those conditions which give rise to boils and carbuncles; and

(5) Tubercular tendency inherited or acquired may lead to this disorder.

Fistula generally begins by formation of an abscess immediately beneath the skin just outside the anus, starting in the cellular tissue, or hair, or sebaceous follicles. The ischio-rectal fossa is a rarer situation. It may begin in the subcutaneous connective tissue of the rectum, and burst into the bowel. It may undermine the rectum in any direction. In phthisical patients it may begin by ulceration of the mucous membrane, in which fæcal matter accumulates and forms a sinus, opening eventually outside the anus. Abscess may originate in the superior pelvi-rectal spaces, and form sinuses extending in every direction. Rectal abscesses may be acute, chronic, or gangrenous. The acute are accompanied by severe constitutional disturbances, the chronic may be evident only from a painless fluctuating swelling, with thin and discoloured skin, or a flat, boggy, crepitating enlargement by the side of the anus. Gangrenous abscess around the rectum is rare, occurs in persons of broken health, and requires very free incision. Rest, fomentation, and leeches may cut short a threatening abscess if seen early. Usually, however, pus forms, and incision is then the only treatment. The longer an abscess is left unopened the greater is the danger of sinus formation. To open small abscesses, place the patient on the side of the swelling, pass the left forefinger into the bowel, and make outward pressure between this and the thumb; with a straight bistoury held perpendicular to the abscess, open it, cutting outwards, and making the incision at right angles to the anus. In severe cases open the abscess from end to end, and from behind forwards. Introduce the forefinger, and break down any secondary cavities, carrying the finger up by the side of the rectum till only one large sac remains. If there is outward burrowing, make a deep incision into the buttock at right angles to the first. Do not cut away sloughs. The cavity is syringed out, and filled with wool soaked in iodoform and vaseline, 20 gr. to $\bar{z}$j. After twenty-four hours this is changed daily, the wool being very lightly carried to the bottom of the cavity, which must *not be stuffed.* Fistula often follows abscess owing to the movement of the part by the action of bowels and sphincters, the presence of much loose areolar tissue and fat, and the unsupported state of vessels near the rectum.

Fistulæ are:—

(1) Complete, having an opening inside and outside the bowel.

(2) Blind external, having no internal opening.

(3) Blind internal, having no external opening.

In almost every case of complete fistula the internal opening is between the two sphincters. Fæces when liquid, passing into complete or blind internal fistulæ, cause great suffering—burning pain often lasting for hours after an action of the bowels. Any fistula may become complex by the formation of many sinuses. As regards the seriousness of a case, the most important point is whether the sinuses are low down in the rectum or surrounding tissue, or high up as the result of abscess in the ischio-rectal fossa, or above the levator ani muscle. In examining fistula use the probe with a very light hand, letting it find its own way.

If operation is not at once desirable, palliative treatment may be tried if:—

(1) There is no internal opening.

(2) The discharge is thin and watery.

There may be more than one internal opening. The internal opening must be felt with the finger, a tortuous course of the sinus often preventing the probe from reaching it.

In horseshoe fistula the sinus generally runs dorsally from one side of the anus to the other. In children fistula is generally due to worms, and is cured by removing them ; a saline iron mixture should be prescribed in addition to the usual vermicide remedies. In adults palliative treatment consists in the application of strong acids, the outer opening of the fisula being first well dilated. Wool twisted upon a piece of stiff wire is passed down the sinus and withdrawn, and then, similarly, wool saturated with the chosen acid. A drainage tube, just large enough to fill the sinus, is inserted, and shortened daily. More than one application of the acid may be needed, and the external opening must be kept dilated. Spontaneous cure of fistula does very rarely occur. Forcible dilatation of the sphincters sometimes leads to cure, but is very uncertain. The patient need only rest for two days after this procedure. When a cutting operation is to be avoided, the elastic ligature may be used if there is only one sinus. For details of this treatment, which is rarely suitable, readers are referred to the author's larger work on *Diseases of the Rectum*.

Operations on Fistula in Ano.—Thirty-six hours before operation an aperient is to be given, and an injection on the morning of the operation. The patient should always be anæsthetised, as the extent of the operation cannot be foreseen, and placed upon the side on which the fistula exists, the buttocks being brought to the edge of the couch, and the knees drawn up. This is the best position for most rectal operations, but with fistula on both sides the lithotomy position should be adopted. A director is passed into the external opening, and if possible through the sinus and internal opening. When, with the finger in the bowel, the point of the director is felt, it is turned towards the anus, and the tissues over it divided with a curved bistoury. If the point of the director cannot be brought out at the anus, owing to the depth of the fistula, it must be passed well through the sinus, and steadied by the left forefinger inserted into the rectum, while a straight knife is run along the groove of the director, cutting towards the bowel until the tissues are severed. This is not an easy operation. Dilation of the sphincters is of great assistance. If there is no internal opening, the point must be found where only mucous membrane intervenes between the point of the probe and the internal finger. If this is the highest point of the sinus, work the point through, and bring it out at the anus. If it be not the highest point of the sinus, gently push the probe farther, and then thrust it through the tissues into the bowel. Unless the sinus is laid open to its whole extent the patient will not be cured. After dividing a fistula from its external to its internal opening, search with the probe for any sinus running up beyond the internal aperture. Search with the finger tip for lateral sinuses from the main track, or for outward burrowing beyond the external opening. There may be a secondary sinus beneath the main one. This is found by scraping away the granulation tissue of the main sinus. When all sinuses have been slit up, the overlapping edges of the skin should be taken off. In cases with much induration draw a straight knife through the dense track of the fistula, and outwards beyond the external opening. When the operation is completed, some fine cotton-wool is packed with a probe into the bottom and every part of the wound. A pad of wool over the wounds, and a T-bandage firmly applied, complete the dressing. In this, as in all rectal operations, strict surgical cleanliness must be observed throughout. In cases where, owing to the depth of the sinus and the density of the tissues, the point of a probe director cannot be got out of the anus, the author's spring scissors and special director are recommended. In operating upon horseshoe fistulæ only one division of the sphincter must be made. Press a director through the external aperture, and on to its point incise the skin in the middle line behind ; push the director through and slit up. Then slit up the lateral sinuses on directors passed in at the external openings, and brought out at the dorsal incision. Most horseshoe fistulæ are dorsal. Incontinence of fæces does not follow operations upon these cases if the internal sphincter is not divided, and if all loose mucous membrane or piles that may be present be thoroughly removed, so as to insure some contraction of the anus. Internal fistulæ are often connected with hæmorrhoids, and sometimes follow suppuration caused by application to these of strong remedies. The internal opening may be detected only with difficulty, appearing as an ulcer which must be searched with the probe. In operating upon women, cut as little as possible.

Incontinence of fæces results from cutting the muscles, particularly the internal sphincter, in more than one place. On one side the sphincters may be divided quite through without harm if the incision is at right angles to the fibres of the muscles. After operation for fistula the bowels should be confined for three days. The wool should be well moistened and removed a little day by day, beginning on the day after operation. After action of the bowels syringe the wound daily with warm antiseptic lotion, and lay a little cotton-wool lightly along the whole track. Fresh sinuses are generally indicated by sudden increase of discharge. Whenever a wound secretes more than its

surface seems to warrant, or pus oozes up on pressure, search diligently for the sinus. Burrowing has certainly commenced. Patients should be encouraged to state when they have pain in or near a healing fistula; this may lead to early detection of small abscess or sinus. The length of time that a recumbent posture is to be maintained depends upon the general health and extent of the wounds. Patients need not be kept long in bed, but should be made to recline upon a sofa. Never, if it can be avoided, operate upon a fistula that is acutely inflamed. A free dependent opening only should be made, and the patient rested till all inflammation has subsided.

Incontinence of Fœces; Causes and Treatment.—Incontinence may result from injudicious operation, even on ordinary fistulæ, or from extensive fistulæ. This distressing symptom is associated with three conditions of sphincter:—

(1) Too deep a cut has been made through both sphincters, and the wound in healing has left a deep sulcus.

(2) The sphincter has been divided in two places, and weak splicing has ensued, the muscles being divided obliquely instead of at right angles to their fibres. Cicatrices are seen and felt, one each side of the bowel.

(3) In women the anal sphincter has a weak point in the perineum, owing to decussation with fibres of the sphincter vaginæ. When this point is incised loss of power results. In these cases little is found on examination, except that the scar shows the fistula to have been perineal.

A single operation may succeed in curing any of these conditions, but many may be necessary, and months may elapse before a satisfactory result is obtained. In most cases, however, free application of the rectal cautery is to be employed. The external and internal sphincters are deeply burned in several places, the old scars cauterised, and the tissue just around the anus burned into. The wounds are to be dressed with wool saturated in olive oil. After-treatment needs great care, daily passage of the finger keeping contraction within due bounds, and soft motions being habitually procured. In the condition first described, where there is a sulcus, the old scar tissue may be cut away, and the wound united by catgut sutures. In the second condition the cautery affords the only hope. In the third condition Lawson Tait's operation on the perineum may be performed, and the flaps turned into the rectum.

Fistula in conjunction with phthisis requires special consideration, particularly as regards the question of operation. *Tubercular fistulæ* are commonly distinguished by the following attributes:—

(1) Fistula begins with a chronic abscess, which is frequently painless;

(2) The external opening is large, ragged, and irregular in form;

(3) The internal opening is large;

(4) The sinuses are soft, and felt as gutters or depressions below the skin;

(5) The skin around the external opening is blue and unhealthy looking;

(6) Discharge is thin and watery, and tubercle bacilli may be found in it;

(7) The buttocks are thin, and the ischio-rectal fossa depressed;

(8) There is an excessive amount of hair, which is long and fine;

(9) The sphincters are weak, little resistance being offered to the introduction of a finger.

Fistula with some or all of these characteristics may be met with in persons:—

(1) The subjects of active tuberculosis;

(2) Who have had hæmoptysis, and still have remains of a cavity or consolidation of apices;

(3) Who have the family history of phthisis, but no direct evidence in themselves, except the fistula.

In the first class only such operation as is necessary to relieve pain will be recommended; in the second, operation with very careful limitation of incision; and in the third, an early and free operation to rid the patient from a possible source of future infection. These patients are generally better subjects for chloroform than for ether. After operation these patients should not be kept strictly on the back or side, as usually after an operation for fistula. They should be allowed up, or in a sitting position, even from the day after operation. Careful feeding, drug, and climatic treatment will help to a favourable result.

Hæmorrhoids.—Piles are generally described as being—

(1) Internal or "bleeding";

(2) External or "blind";

and the distinction is as a rule well marked, although internal hæmorrhoids may so prolapse as to be mostly external, and external piles may encroach upon the mucous membrane within the anus. External piles are the less important. They are of two kinds: the first being hypertrophies of skin, exaggerations of the surface of the anus; and the second, venous tumours rounded or elongated, situated at the verge of the anus, or passing up into the bowel. External piles cannot be reduced within the anus. Internal piles, if protruded, can be so reduced by gentle pressure, and thus in doubtful cases a diagnosis may be made as to which of the two kinds, or whether both are present. When both kinds are present the case must be regarded as one of internal piles. External hæmorrhoids are so common that few persons reach middle age without suffering from them in greater or less degree. So great a variety of causes helps to their formation that no class of persons is exempt, but those suffer

most perhaps who are prone to rich living, excessive smoking, sedentary occupations, or violent prolonged exercise. Straining at stool, the use of cheap printed paper as a detergent, constipation, and pregnancy are some of the factors very commonly present. Either variety of external pile—the simple skin hypertrophy and the varicose clot-containing vein—is liable to acute attacks of inflammation. Extra eating and drinking are often the exciting cause of these. A sensation of fulness and pulsation in the anus, followed by itching of a most annoying nature, is the first symptom. The anus is a little swollen and tender, and there may be a stain of blood when the bowels act. All this may pass off with simple treatment, but if neglected will precede a more severe attack. One common mistake, to suppose the piles internal, and attempt their reduction, must be carefully guarded against. The necessary treatment consists in spare diet, rest, reduction of smoking, an aperient pill, and the local application of a sedative and astringent ointment or lotion. Common and efficient prescriptions are—Bismuth subnitr. ʒij., Hyd. subchlor. ʒj., Cocaine, gr. iv., vaseline ad. ℥j.; or Liq. plumbi. subacet. ℥j., Liq. opii. sedativ. ℥ss. A tumbler of Vichy water or Carlsbad salts, taken on rising, is often a useful adjunct. If, through delay in treatment, more acute inflammation sets in, the piles become much increased in size, œdematous, or shiny, and very painful if touched. By irritation they cause spasm of the sphincter and levator-ani muscles, giving rise to acute pain just as the patient is falling off to sleep. He is generally feverish and constipated. Warm applications are in this state generally better than cold, but sometimes ice gives much relief. Treatment as recommended above will probably cure the attack, but if desired, operation may be at once undertaken. It is to be recommended—

(1) For the saving of time;
(2) To prevent recurrence;
(3) If the pain is great, and unrelieved by palliatives.

In the case of the hypertrophied skin tumours, operation consists in removing them with scissors, and may be done under gas. Care must be taken not to remove enough skin to induce subsequent contraction. The venous piles are to be pinched up between finger and thumb, and the basis transfixed with a curved bistoury, which is made to cut out Pressure extrudes the clot, and fine cotton-wool is then placed at the bottom of the sac. The incision is made in the direction of the radiating folds of the anus. If unoperated on these tumours may form cutaneous flaps, the blood in them becoming absorbed.

Internal Hœmorrhoids.—All causes of external piles tend to the production, too, of internal, but these may also represent a family idiosyncrasy. Pregnancy, which often causes external piles that pass away after labour, has a different relationship to internal hæmorrhoids which appear after parturition, when the parts are relaxed and involution is taking place. Internal piles have been attributed to anatomical peculiarities of the superior hæmorrhoidal veins, but their diverse structure is against this view. They may be—

(1) Capillary;
(2) Arterial; or
(3) Venous.

The first are small, florid, raspberry-like looking vascular areas on the mucous membrane, with a granular surface that bleeds on the slightest touch. They consist of hypertrophied capillary vessels, and spongy connective tissue, and closely resemble arterial nævi. After a long time, or after strong applications, they lose their granular appearance, and may remain dormant for longer or shorter periods, when they grow again. Arterial hæmorrhoids are sessile or slightly pedunculated tumours, with glistening or slightly granular surface, and having an artery, often as large as the radial, entering the upper part. They consist of numerous freely anastomosing vessels, tortuous, and sometimes dilated into pouches, and a stroma of connective tissue.

In the third variety of internal piles veins predominate. The tumours are often as large as a hen's egg, of bluish or livid colour, with a smooth or pseudo-cutaneous surface. Ordinary piles of long standing, causing discharge rather than bleeding, have been spoken of as "white piles." Partial prolapse of the mucous and submucous membrane of the rectum much resembles, but is to be distinguished from piles. There is no definite tumour, and no hypertrophy of vessels.

Capillary piles can be diagnosed only by symptoms and ocular inspection; they cannot be felt. Pains in the back, in the spermatic cord and testicles, and small daily losses of blood are commonly met. It is this form of the disease that may be benefited by application of carbolic or fuming nitric acid. The treatment is uncertain, even with capillary piles, and never to be used with the arterial and venous forms. Powerful astringents, as liq. hamamelis, twice daily, or suppositories of sulphate of iron, are similarly sometimes effective with capillary piles. Arterial and venous piles have many symptoms in common, and the suffering they cause depends more upon the condition of the tumour, as regards inflammation or ulceration, than upon its exact histological nature. In the early stages of the affection the piles come down at stool and bleed, returning within the sphincter after the bowels have acted or when the patient rises, or at least upon his lying down and voluntarily retracting. Later, pressure is needed. On their return they come outside the

sphincter on the slightest exertion, discharge mucus, bleed independently of action of the bowels, and cause a constant sense of weight above the anus, the patient feeling as if the bowels were never properly relieved. Cutaneous hypertrophies are generally present around the anus. These are irritated by the discharge, becoming inflamed and very tender. Sometimes a number of polypoid growths are studded over the mucous membrane. Arterial and venous piles can be felt on examination as tumours bulging from the rectal wall with well-marked sulci between them. If scratched they bleed freely. Arterial piles are less dependent upon constitutional causes, and have less tendency to prolapse than the venous.

Treatment. — Palliative treatment has little effect upon arterial piles. With venous piles it may materially alleviate, if not cure, the disease. Such treatment consists in regulating the diet and mode of life, careful regulations of the bowels, the use of cold water injection after their action, and the application of suitable astringent ointments. Advanced cases need operative treatment.

Treatment of Protruding Piles.—If the piles have just come down the patient should lie prone with pillow under the pelvis. Wool saturated with 20 per cent cocaine solution is left on the piles for ten minutes; the finger is then passed up the bowel, while gentle attempts are made by pressure with the other hand to empty the piles of blood. If this fails a bladder of ice should be placed over the part for one hour, and then taxis again tried. This again failing, apply cocaine ointment and make no further attempt at reduction. If the piles have been down for some time and are inflamed or even sloughing, and cannot be returned, they must be treated with ointment and fomentations. If they can be returned, but immediately prolapse again, the same treatment is to be used. It causes profuse suppuration with speedy separation of the sloughs. Stimulants and tonics are often necessary in such cases. In pregnancy piles should be operated on only if there is much loss of blood and great suffering. The recumbent position is to be kept longer than with other cases. In women the subjects of uterine displacement this must be remedied before any operation for piles. Complicated cases of disease of rectum, uterus, and bladder are common and most difficult to remedy. When piles are causing serious loss of blood every day's delay in operation is to be deplored, and no consideration should lead to postponement of the only measure which can effectually relieve the drain upon the strength.

Operations for Internal Hæmorrhoids.— Thirty-six hours before operation, two pills are to be given, composed of: Pil. hydrarg. gr. j., Pil. col. et hyoscy. gr. iv. When the piles are venous, and in some plethoric patients, a longer course of preparatory treatment is desirable; while in the very anæmic less drastic purges are better. On the morning of operation an enema is given. The patient is placed upon the right side at the edge of the bed. The assistant stands with his back towards the patient's head, raising the upper buttock with his right hand, and with the right elbow hooked over the patient's pelvis, thus controlling the position. There are several operations from which to choose in treating hæmorrhoids. Before describing that by ligature, which we recommend in the vast majority of cases, we will briefly allude to others which may in certain cases be preferred.

(1) Excision by Knife or Scissors.—This must only be done when the piles are not large, vascular, or numerous. The so-called "white pile," partially prolapsed mucous membrane, and the single perineal pile, so frequently found in women, are conveniently operated upon in this way. The sphincters are first gently, but fully dilated. The pile is seized at the base by a vulsellum and cut off. The vulsellum is kept in place till any spurting vessels have been clipped and twisted; rarely more than two require attention. When bleeding has ceased, cotton-wool soaked in solution of tannin and water (equal parts) is placed within the anus as high as the scissors have cut.

(2) The ecraseur formerly often used has now no recommendation in operating for piles. Its use is followed by intense and prolonged pain.

(3) Application of Strong Acids.—This must be reserved for the treatment of small granular piles or patches of villous bleeding mucous membrane. Eucaine (4 per cent) is applied, and the area to be treated surrounded with wool soaked in bicarbonate of soda. The surface is then dried and the acid applied several times with a small wooden brush. The parts are then washed, oiled, and returned within the sphincters.

(4) Injection of Carbolic Acid.—The advantages of this method are that confinement in bed is not necessary, and it can be employed without a general anæsthetic. Its great drawback is that it is far more dangerous than any other method of operating. Dangerous hæmorrhage, embolism of the liver, and many deaths have followed injection. The injections are made with a hypodermic syringe, and may have to be repeated after a fortnight. Injection should never be performed when there are many piles, or when a pile is one which cannot be completely returned within the sphincters. Injection may stop bleeding and diminish prolapse, but it is not to be regarded as a cure.

(5) Cauterisation.—There is the same objection to this as to the injection method. A slough or inflammation is produced, the extent

of which is beyond control, in the interior of a vascular tumour not cut off from the general circulation. The cautery must be used only in the case of a capillary pile; the point is to be freely applied all over the bleeding area, but not to penetrate deeply the wall of the bowel. There is no advantage in employing a galvanic cautery. Paquelin's is simple and preferable.

(6) Removal by clamp and scissors, bleeding being arrested by the heated iron. This "clamp and cautery" operation is six times as dangerous as the ligature, leaves great pain, and a greater likelihood of secondary hæmorrhage, and there is more often troublesome subsequent contraction.

(7) Dilation of the Sphincter Muscles.—In some cases when the piles on protrusion are tightly gripped by the sphincters, become swollen, and perhaps bleed freely, speedy relief and even a cure may result from mere dilatation of the sphincters, gently but fully carried out. The method is of little value in other cases unless the hæmorrhoids have existed only a short time. The operation of dilatation is of great use for the mere relief of pain in cases where for any reason a more radical proceeding is to be avoided.

(8) Treatment by Crushing.—This method is better than the "clamp and cautery." It may be used when the piles are small and not more than three. It should not be used for inflamed piles nor in cases of anæmia. The author's screw-crusher is recommended as giving less chance of recurrent hæmorrhage, and details of the operation are given in his larger work. The operation is safe in chosen cases, but is rarely to be preferred to the ligature.

(9) Whitehead's Operation. — Theoretically, this proceeding, which is based upon complete removal of the pile-bearing area, is excellent. Practically it is unnecessarily extensive, and one of the worst operations for piles. Moreover, recurrence is more likely after this than after operation by the ligature, and the proceeding requires about six times as long as the latter. Three evil results that have frequently followed this operation confirm our opinion against it—

(*a*) Anal stricture.

(*b*) Loss of sensation in and power over the anus.

(*c*) The mucous membrane, stitched down to the skin around the anus, adheres outside it. The mucous membrane is exposed to irritation, discharges mucus, and bleeds. We do not give a detailed account of the operation, because its performance is not recommended.

In those very rare cases in which the entire circumference of the rectum is to be excised for piles, Mr. Thelwall Thomas's modification of Whitehead's operation is the quickest and safest operation. The chief point is in the method of suturing the bases of the piles, for description of which the reader is referred to a larger work on the subject.

(10) Operation by Ligature. — This is, as above stated, in our opinion the safest and best operation for the great majority of cases of hæmorrhoids. It is begun by complete dilatation of the sphincters. The hæmorrhoids are drawn down one by one, and with sharp scissors separated from their connection with the muscular and submucous tissues. The cut is made in the white sulcus where skin and mucous membrane meet, and carried up the bowel parallel to its axis till the pile is left connected by an isthmus of vessels and mucous membrane only. As the larger vessels come from above and enter the pile just beneath the mucous membrane they are unaffected by this incision. A strong silk ligature is placed in the deep groove now made and tightly tied with three knots, while the assistant draws the pile well out. If the pile be very large a portion may be cut off, enough being left to guard against the slipping of the ligature. A strip of wool saturated with iodoform ointment is passed into the bowel, and a pad of wool over the anus firmly secured by a tight T-bandage. It is most important when there are many piles, that the base of the ligature should be as narrow as is consistent with non-division of the chief arterial supply; otherwise too great a narrowing of the rectum is produced. In 5000 cases of this operation we have had only 5 fatal results. Other surgeons show an equally satisfactory experience of the procedure.

After-treatment.—The bowels should be confined for three days. A hypodermic injection of morphia is given just after operation, and repeated if necessary for pain during the first twenty-four hours.

In very bad cases and in delicate persons the bowels should not be allowed to act for even four days or more. Diet should be light and such as leaves little residue. There must be no alcohol, and complete rest in bed. When retention of urine occurs the patient should kneel in bed with warm flannels over the pubes. After twenty-four hours the pad of wool is changed, and the strip inside the bowel removed after forty-eight. A pad soaked in sanitas is then kept applied. On the third night, Pil. hydrarg. gr. ij., Pil. col. c. hyoscy. gr. viij., are administered. When the bowels act the patient, unless anæmic or ill, may get out of bed. There is often pain and a little blood with the first motion. If there is much straining the bowel is to be examined for the presence of hard fæces, which must be broken up with the finger, an enema of warm gruel and olive oil being then given. After the first action of the bowels fomentation is to be applied. A daily action by suitable mild laxatives is now to be secured. The wounds are dressed daily with antiseptic lotions or ointments suitable to the condition

present. A weak antiseptic solution is syringed in after every action of the bowels. The dressing consists of a small strip of wool for each wound, carrying the lotion or ointment prescribed. After the first week the finger is passed daily to make sure that no contraction results The patient may now get up daily to lie for a few hours on a sofa. He must not remain long in the erect position, for, though convalescent, his wounds are not yet healed. Rest must be insisted upon until the bowel is felt to be healed. Ligatures usually separate about the eighth day.

The following conditions are often found in conjunction with hæmorrhoids :—

(1) Fissure.—A small painful ulcer is often present in cases of piles, and is commonly overlooked because a careful examination is necessary to find it, hidden as it is by one of the hæmorrhoidal tumours. Pain on defæcation or long after the bowels have acted should always lead to suspicion of fissure.

(2) Fistula is not so common a complication, but is sometimes present, and needs care for detection if it be of the blind internal kind.

(3) Polypus and polypoid growths are sometimes found with piles. They must be removed at the operation, or they may prevent healing of the wounds.

(4) Impaction of fæces is sometimes found when the operation for piles is begun. The mass must be broken up and washed away before the surgeon proceeds further.

(5) Lax Condition of Sphincters. — In old persons and in those who have had large venous piles for a long time this conditiom may be met. In such cases it is not advisable to perform the preliminary dilatation.

(6) Malignant Disease.—In examining a case of hæmorrhoids, the surgeon should never omit to pass his finger well into the bowel before withdrawing it, and thus be sure that there is no stricture, ulceration, or malignant growth.

Complications that may follow the operation for Hæmorrhoids—

(1) Loss of Power in the Sphincters.—When large piles have been removed a sense of weakness and loss of power over the anus may be left. The piles acted as a plug, and at the same time caused, from their prolapsing, a weakening of the sphincters. Moreover, the change in the mucous membrane effected by operation brings about a dulness of appreciation when fæces approach the external orifice. Thus the patient may be unable to prevent discharge of wind or motion. If the anus is bathed night and morning with cold water, and an ounce of some stimulating lotion be daily injected into the rectum, the proper power soon returns.

(2) Impaction of Fæces.—Although no fæces may be seen at the operation, large masses may be retained in the large intestines in any case of rectal disease. Delayed healing is often due to such descending masses.

(3) Contraction of Anus.—This is especially likely if the patient is allowed to get up too soon. It usually affects the mucous membrane only, and is cured by daily passage of the finger or bougies. In rare cases, when there has been long healing, or where extensive removal of the wall of the rectum was necessary, a tight hour-glass contraction is found. Constitutional as well as mechanical treatment is generally necessary, as such a condition occurs almost only in weak and unhealthy subjects. The patients are generally women, and there is often some uterine condition which needs care to permit of cure of the stricture in the bowel.

(4) Abscess and fistula should be looked for when suppuration is more profuse than is consistent with the healing of the wound, or separation of sloughs.

(5) Bubo or pelvic suppuration may arise in delicate patients.

(6) Hæmorrhage may be primary, recurrent, or secondary. The first is very rare if the operation is properly done. If no important vessel can be discovered bleeding, a little subsulphate of iron wool is to be introduced into the rectum, pressure applied, and an ice-bag placed over the exposed buttocks.

Recurrent hæmorrhage after many hours, or even a day, is more serious. If the treatment just described does not stop the bleeding, and especially if the patient feels distended, there is probably internal hæmorrhage. Prompt action is necessary. Remove the bandage, pass a finger into the bowel, and tell the patient to bear down. Dark blood and clots pour out. Syringe the rectum with cold water. If the bleeding still continues, pass the finger again and find the spot where pressure controls the hæmorrhage. Ether is now to be administered, the nurse continuing the pressure upon the bleeding spot, if another medical man is not at hand. When the patient is properly anæsthetised the nurse must continue the administration while the surgeon pulls down the bowel with a vulsellum, and picks up the bleeding vessel. If it cannot be found clips must be left on the mucous membrane at the place where pressure controls the loss. If this too is ineffectual, the bowel is to be plugged. A No. 12 gum-elastic catheter with the end cut off is passed up the bowel for six inches. A cone-shaped sponge, with strong silk ligature passed through its apex, is passed four inches up the bowel, the two ends of the ligature hanging out. Then the whole rectum below the sponge is to be carefully packed with antiseptic wool or gauze. Now pull the sponge downwards by the ligature, at the same time pushing the stuffing up. In this way the sponge is distended, and no bleeding can possibly take place. The plug is left in about one week, and the patient kept if neces-

sary under the influence of opium. Wind passes by the catheter.

Secondary hæmorrhage may come on at the time of separation of the sloughs. It generally occurs only in elderly subjects of broken-down constitutions. Generally the tissues are too rotten for application of a ligature. Treatment as just described must at once be instituted. Afterwards the buttock and lower part of the bowels are to be kept cool, and a hot bottle put near the head if the patient is faint. Careful nourishment and after-treatment are needed. In women, hæmorrhage from the rectum may be controlled by the fingers in the vagina, making backward pressure against the sacrum.

Procidentia Recti.—Procidentia and prolapse are often confused. Procidentia is a descent of the whole circumference of the rectum, prolapsus is a protrusion outside the anus of a portion or portions of the mucous membrane. Internal hæmorrhoids, when they come outside the anus, are said to be prolapsed.

Procidentia may take place in three ways :—

(1) The entire circumference of the mucous membrane, or all the coats of the rectum, appear outside the anus.

(2) The upper part of the rectum descends through the lower, and appears externally.

(3) The upper part of the rectum descends through the lower part, but does not appear outside.

Constipation is usually a symptom in children, but in the old a diarrhœa with discharge of mucus is more common. In old and bad cases there is more or less incontinence of fæces.

In the third variety there is obstinate constipation, a sensation of burning and fulness with tenesmus, straining, difficulty in defæcation, and occasional discharges of blood and mucus. These symptoms should lead to an examination of the bowel with the patient standing up. The finger passing up close to the front or back wall of the bowel meets an obstruction, then, slightly withdrawn, it will find in the centre of the bowel the orifice of the descending portion. This condition occurs chiefly in the old. The other forms of procidentia are most common in children. Reason for this may be found in the length of the mesentery, though worms, stone in the bladder, phimosis, and polypus recti are also sometimes to blame. Occasionally the cause is indiscernible. When a large portion of the bowel is down there is much difficulty in returning it. Gentle taxis, and pressing up a large flexible bougie, generally succeed. If there is much swelling, rest must be enforced with the buttocks well raised ; after a few days the mass may be returned. Constant application of ice must not be made, or serious sloughing may ensue. With children palliative treatment generally succeeds in curing the condition. Any source of irritation is to be removed, and the general health attended to. Sitting and straining at stool are forbidden, the motions being passed while the child lies at the edge of the bed, or stands up, one buttock being drawn aside so as to tighten the anal orifice. Afterwards the protruded part is washed with cold water, and thoroughly sponged with weak carbolic or hamamelis. The bowel is then returned by gentle pressure, and the child kept lying upon its face for half an hour. Mild measures failing, the application of strong nitric acid is the best remedy. An anæsthetic is given, and the protrusion well dried. The acid is applied all over it, and then after being oiled the parts are returned, and the rectum stuffed with wool. A pad is applied outside the anus, and the buttocks are strapped together. The bowels are confined for four days. Then castor oil is given, the plug comes away, and the rectum does not descend. Sometimes more than one application of the acid is necessary.

Procidentia in adults is cured with much greater difficulty. For the first and second varieties, the best mode of treatment is free application of the actual cautery, assisted, if necessary, by opening the abdomen, and pulling up the gut from within. For the third variety, pulling up the gut from within, and fixing it to the abdominal wall, is the safest and best mode of treatment. The cautery is used in the following manner :—

The patient is anæsthetised, preferably with ether ; the part, if not quite down, is drawn fully out of the anus by a vulsellum. With the iron cautery at a dull red heat, make four longitudinal stripes from the base to the apex of the procidentia, taking care not to burn so deeply towards the apex as at the base. If the procidentia is very large, six stripes may be made. The intestine is then oiled and returned within the anus. The sphincters are partially divided on both sides of the anus by a sawing motion of the hot iron, and a small portion of the oiled wool is inserted. The patient must be kept in bed, and the motions passed lying down. If the wounds are not healed in a month he must continue the recumbent position for another two weeks, by which time it rarely happens that all are not healed. After he is up and about, motions should be passed lying down.

Polypus Recti and Polypoid Growths.—By polypi we mean pedunculated growths attached to the mucous membrane of the rectum, and generally situated not less than one inch from the anus. They may be found two inches up the bowel, but rarely more than that. Generally they grow from the dorsal portion of the rectum. The two common kinds are—

(1) *Mucous or Soft Polypi.*—These are small vascular tumours with pedicle often two inches long. They resemble a half-ripe mulberry. These polypi may bleed very freely. They are true adenomata.

(2) *Fibrous polypi*, which originate from the submucous connective tissue. These may be very hard and have pedicles always attached above the sphincters. In adults polypi are generally neither very hard nor very soft. They are sometimes cystic.

Symptoms.—In children there is frequent desire to go to stool, tenesmus, occasional bleeding with discharge of mucus, and a fleshy mass protrudes when the bowels are acting. When situated high up polypi may be dangerous by causing intussusception. The peduncle is slender, and probably polypi are at times spontaneously cured by separating when a hard motion is passed. In adults there is often the history that without previous discomfort a substance is discovered to protrude when the patient is at stool. This is characteristic. Until the peduncle is long enough to allow the polypus to be protruded or grasped by the sphincters, there is little or no inconvenience, and the onset of the disease therefore appears quite sudden.

This is different from the history of a case of hæmorrhoids. A certain diagnosis is made by passing the finger to its fullest extent, and sweeping round the rectal surface as it is withdrawn. The disease may be mistaken for internal piles, procidentia, or dysentery. An examination after an injection will decide against the first two, and the patient's general condition and nature of his motions against the last.

The only treatment to be recommended is removal of the growth. The peduncle is to be seized close to its base with torsion forceps, and gently twisted till the polypus comes away. There is no hæmorrhage or pain, and no necessity for resting more than one day. If the polypus be large it should be drawn down, and a threaded needle passed through a small piece of the mucous membrane at the base of the pedicle. A knot is tied, and the pedicle then surrounded with the ligature tightly tied. The polypus is then cut off, not too near the ligature. In such a case the patient is to be kept at rest until the ligature separates, and the bowels confined for three days. *Disseminated polypi* constitute a rare disease peculiar to certain families, several members of the same family being affected. The symptoms resemble those of the soft polypus in children. The treatment is of uncertain result. Attempts to relieve by means of astringent injections are futile, and the most efficient treatment is to dilate the sphincters, under an anæsthetic, and with the finger and a Volkmann's spoon to scrape away the polypi as high as can be reached. Tenesmus and rectal discomforts are relieved in this way, but of course the polypi situated in the upper part of the sigmoid flexure and colon are unaffected, so loose motions of mucous and blood continue. The scraping may have to be repeated. There is sharp bleeding, which must be arrested by hot injections. Complications sometimes arising from polypus are abscess, ulcer, fissure, and fistula. If with history of polypus none can be felt, the patient must be examined just after an administration of an enema.

Polypoid growths are small growths not absolutely pedunculated protruding from the rectal mucous membrane. They rarely come outside the anus. There are two kinds, both to be distinguished from warts, which chiefly affect the outside of the anus. One kind are little tags of mucous membrane, never more than one inch long, generally situated on a small pile, or at the upper part of a fissure. The other kind are hard and nipple-like, with broad base and firm-pointed apex. They are composed of dense fibrous tissue. Discharge of mucus, or a moist watery condition of the anus, is the symptom for which such growths usually lead patients to consultation. Examination with finger and speculum reveals these growths, which are to be removed with the scissors.

Warts around the anus are as warts elsewhere. They are best removed by applying fuming nitric acid to each individually, and scraping them off, applying acid again to their bases.

Pruritus Ani or Painful Itching.—This is a distressing and intractable affection, but is always curable if the patient will patiently and persistently follow out treatment. The general causes are too free living, hereditary predisposition, and various disorders, of which gout is the most frequent. Particular articles of diet affect some persons—lobster, for instance, invariably causing pruritus in certain subjects. Spirits, coffee, and excessive smoking often excite the complaint. Occasionally it must be considered as a pure neurosis. Local causes are constipation, piles, polypi, fistula, and vaginal discharges. The itching is usually worse at night, but in excitable persons often comes on after exercise, or on entering a warm room.

Usually the affected part shows a distinct rash, or is dry, rugose, or bright red. Sometimes there are a number of minute scales forming irregular rings upon it. The most characteristic appearance is loss of the natural pigment of the part. General treatment must be appropriate to the cause, and is of the highest importance. Locally, irritation is best relieved by strong alkaline solutions frequently applied. Light diet, exercise, and mineral waters play a prominent part in the treatment of most cases, and nervous people require sedatives, sometimes in large doses, at bedtime. Opium is to be avoided. Ointments, lotions, and powders may have to be tried, and before their application the part is to be washed with Castile soap, or oatmeal and water. In obstinate, old-standing cases the parts should be rubbed with solution of nitrate of silver, 40 grains to the ounce of water. When the irritation is so

great that the sufferer is worn out for want of rest, a bone plug may be inserted into the anus at night. It is shaped like the teat of a feeding-bottle, and has a circular shield to prevent slipping. In some cases, when there is no discoverable cause, benefit accrues from dilating the sphincters.

Fissure and Painful Irritable Ulcer of the Rectum.—This small affection wears out a patient's health and strength in a remarkable manner, if, through motives of delicacy, for instance, it is allowed to go on a long time. Yet it is very easily cured. Ulcer, or fissure, which is only a small ulcer in a particular position, may be caused by constipation, polypi, syphilis, or congenital narrowness of the anal orifice. It is commonly situated along the internal or external sphincter. Ulcers and fissures vary in depth and size. They may be simple wounds, or inflamed, callous, indurated, or even with a sloughy base. The most usual position is dorsal. Usually the patient believes himself to have piles, complaining of discharge of blood and matter, and swelling and pain at stool. The pain at stool may be excruciating, leaving the sufferer to postpone action of the bowels, and thus increasing the pain eventually produced by passage of hard, desiccated fæces. Often pain continues long after a motion, and sometimes does not begin till some minutes after. The great mobility of the external sphincter, and its free and easily exposed nerve-supply, account for the severity of the pain. Retention of urine, pain in the back, and numbness down the legs may be caused by fissure. Examination for fissure is conducted in the manner described as usually to be adopted for rectal examination. A polypoid growth will often be seen at the upper part of the fissure. A speculum may be needed for detection of an ulcer, though the educated finger distinguishes the spot at once, for it feels like the internal aperture of a fistula, but with harder edges, and without elevation above the level of the mucous membrane. In children and young persons, unless a polypus or polypoid growth is present, cure can be obtained without operation. Rest, mild laxatives, mercurial ointment, and cleanliness are all that is necessary. After action of the bowels an opiate injection is to be used. If there is great spasm of the sphincter, belladonna ointment is to be smeared around the anus over the sphincter. When a fissure is anterior or lateral, it can be cured without operation. When it is dorsal, and there is no spasm or hypertrophy of sphincter, it can also be so cured. When the fissure is dorsal, with spasm or hypertrophy of the sphincter, palliative treatment is not enough. Division, or very free dilatation of the sphincters, is then necessary. The external sphincter must be completely divided, the internal only stretched. The incision should begin a little above the upper end of the fissure, and end a little below the outer end. Its depth should be not less than half an inch. Polypoid growths, and swollen, inflamed skin, if present, are to be removed with scissors. A small piece of cotton-wool is placed in the wound for forty-eight hours, and the patient kept in bed till the wound is healed, after-treatment being as with fistula cases. Complete dilatation is effected by pressing and pulling with the thumbs till the sphincters, previously hard, feel like putty. It takes five minutes to do this thoroughly. All ulcers about the internal sphincter are best treated by incision; old, large, indurated fissures also require this treatment, and those complicated by piles or fistula. Simple fissures about the external sphincter, and those in the old, phthisical, and broken-down in health, and in children, if the operation is necessary, should be cured by dilatation.

Neuralgia and Hysteria of the Rectum.—This term is applied to a painful condition of rectum, when nothing can be found to cause the symptoms. Many slight lesions are easily overlooked, and the absence of these must be certainly ascertained before any case is christened neuralgia or hysteria.

(1) Very slight erosion or inflammation of a spot in the rectum, high up or low down, may cause great pain, and yet be found only with utmost difficulty.

(2) Fissures and irritable ulcers may give rise to nervous sensations, even after they have healed. Perhaps a nerve filament is implicated in the cicatrix. When no local cause is present the symptoms are—

(*a*) Reflex, caused by lesion elsewhere.

(*b*) Actual neuralgia, or hysteria.

These cases do occur, though not commonly. Sufferers from these obscure affections are generally healthy people with no other trouble. They complain of short, stabbing pain, unrelated to action of the bowels, with no apparent cause, and at times very intense. Others have a sense of fulness and burning, unrelieved by purgation or washing out the bowel. Others are frequently obliged to go to stool, where they get no relief, while others are burdened with a constant sense of the presence of the rectum. In such cases, when the most careful examination reveals no local or distant cause for the symptoms, these must be regarded as neuralgic or hysterical. Conditions with which such symptoms are associated are—

(1) Congestion of the rectal vessels.

(2) Uterine, urinary organ, and prostatic disease.

(3) Locomotor ataxy.

(4) Mania.

(5) Epilepsy.

When none of these exist, and there is no local cause, we are confronted with a true neuralgic or else a hysterical rectum. Pos-

sibly neuritis of rectal nerves may explain the pain.

Persistent Constipation.—The surgical aspect of this complaint may here be appropriately considered :—

(1) Spasm with hypertrophy of sphincters is not at all an uncommon cause of persistent constipation. It may be first induced by an attack of constipation, which it then itself renders persistent. The original attack may arise in many ways, very commonly from irregularity in attending to action of the bowels. The rectum and colon are over-distended, and cannot act forcibly. The fæces become hard and dry, and the muscular tissue of the bowel weakened, perhaps poisoned. Straining, with little or no result, follows. From this, and the irritation of the fæces, spasm and hypertrophy mainly result. In examining such a case it is found that no dilatation of the anus follows upon the patient's bearing down. On introduction of the finger the sphincters are felt to be tight and broad.

(2) Congenital narrowness of the anal orifice is another important cause of chronic constipation.

(3) The condition may result from atony combined with loss of muscular sense.

(4) The third variety of procidentia may tend to severe constipation.

(5) Malformation of the uterus and enlargement of the prostate may be the cause, or the constipation may be a reflex result of renal calculus.

(6) Irreducible hernia.

Treatment.—As an adjunct to all methods of treatment a copious enema is to be given; a tube should be used not shorter than six inches, with a calibre not less than one-third of an inch, and open at the top. In bad cases a much longer tube is to be employed. To pass this the left forefinger is introduced into the rectum, and the tube slid along it beyond the promontory into the sigmoid flexure. Now inject water, simultaneously pushing the tube up the bowel. As much water is to be injected as can be borne without pain. In such cases forcible dilatation of the anus is often necessary for a cure, or in less severe cases daily passage of bougies in increasing size. For the congenital cases forcible dilatation or division of the sphincters, followed by daily use of bougies, is the only satisfactory treatment. In the old or feeble a systematic course of galvanism, one pole in the rectum, and the other moved about over the surface of the abdomen, is to be recommended. Massage is useful. When the subject of constipation has a lax and pendulous abdomen a suitable belt materially assists in curing the complaint. Glycerine injections are of service only so long as they act as a local irritant. When the rectum becomes accustomed to them they are of no more service than warm water.

Impaction and Concretions.—These may follow a single attack of constipation. Their presence is generally marked by the occurrence of diarrhœa. The cause is nearly always loss of power in the muscular coat of the rectum. The accumulation takes place in the pouch just above the internal sphincter. There is always spasm of the sphincter. The symptoms are obscure, and the malady often misinterpreted. A common error is to treat the patient for diarrhœa with tenesmus. Severe pains may be experienced in the right lumbar and left inguinal regions. Dyspepsia, irritability of temper, nervousness, and despondency, morning vomiting, distaste for food, thirst, and frequent liquid motions without relief, are common symptoms of impaction. On palpating the abdomen tumours may be felt in the transverse colon or sigmoid flexure. The anus is nipple-shaped, and the sphincters are tightly contracted, whilst the finger, introduced with difficulty, meets a ball of hardened clayey fæces. When the tumour is high up careful palpation proves it to differ in size and shape at different times, and to be irregularly soft with a doughy feeling.

Treatment.—This is begun by thoroughly breaking up the ball of fæces. An anæsthetic is necessary, for the sphincters are to be dilated, and the mass broken up with a lithotomy scoop. Injection of soap and water, oil, and fresh ox-gall will then fetch away enormous quantities of fæces. Care is required that no reaccumulation takes place, as it is a considerable time before the rectum recovers power after great distension.

Concretions in the bowel are rarer than impactions, and when they exist often give rise to the latter. They are usually formed round some foreign body, and are cylindrical in shape. They consist of animal and vegetable fibres matted round a central nucleus, which may be of various material. Concretions must be removed after dilatation of the sphincters under an anæsthetic; they are too tenacious to be broken up like an impaction.

Ulceration of the Rectum.—Ulceration extending above, or entirely situated above, the internal sphincter is not very uncommon. In the early stages it is amenable to treatment, but, unfortunately, this is rarely offered, the symptoms being obscure and insidious, and the suffering at first so slight that the patient deceives himself. The chief varieties are—

(1) Tubercular.
(2) Dysenteric.
(3) Syphilitic.
(4) Traumatic.

In most cases the history alone indicates the nature of the ulceration, and too much reliance must not be placed upon its character to the touch.

Symptoms.—The earliest is morning diarrhœa

of a peculiar character. The patient on rising immediately feels urgent desire to go to stool. Wind, a little loose motion, and some discharge are passed. The last resembles "coffee grounds," or is like white of egg, or is more rarely pus. The patient has tenesmus, is not relieved, and before long again has to seek the closet. This time lumpy motions smeared with blood are probably passed. After breakfast probably the bowels act again, and he is then fairly comfortable for the day, only an occasional disagreeable sensation reminding him that there is something wrong with the bowel. Mucus or muco-pus may exude from the anus. After some months there is burning pain after an evacuation, and increase in the discharge, which is more largely pus. The pain is induced by walking and standing. Diarrhœa appears now in the evening as well as in the morning, and the health begins to suffer. Anomalous pains are felt in the back, hips, legs, and perhaps penis. Alternating attacks of diarrhœa and constipation may now occur, and the diarrhœa is alternated with severe colicky pain, faintness, or sickness. Attacks of a low form of peritonitis occur. There is tympanitis and tenderness on pressure at one or more spots on the abdomen.

Examining such a case per rectum, the condition found varies according to the stage of the disease. Early, ulcers may be felt within two inches from the anus, with raised hard edge, painful, and readily bleeding. Then there may be lumps, either gummata or enlarged glands. In this stage the disease is often curable. Later there is great thickness of mucous membrane, and outside the anus are swollen tender flaps of skin. So definite is the outside appearance in advanced cases that one glance tells an expert the presence of cancer or severe ulceration. Stricture, and probably fistula, are now present. Perforation into the bladder, vagina, or peritoneum may take place. The patient's state resembles that of a sufferer from malignant disease, and no remedy short of colotomy offers much chance of even temporarily prolonging life. Relief is possible, but cure scarcely.

Palliative Treatment.—Rest in the recumbent position and a fluid diet, consisting mainly of milk, vastly improve these patients. Locally, various sedative and astringent ointments are to be used. Dysenteric cases are treated with large doses of pulv. ipecac. co. in addition, and injections of silver nitrate 20-40 grs. to three pints of water. Syphilitic subjects require a thorough course of mercury if seen early. In all bad cases inguinal colotomy is the best means of relief.

Stricture of the Rectum.—All the different kinds of ulceration may result in stricture; ulceration with stricture also results from improper operations upon the rectum. Stricture without ulceration is somewhat uncommon. It may result from chronic inflammation caused by repeated passage of hard, dry fæces. It has been seen after frequent, perhaps rough use of an enema tube. Bruising of the bowel after a long delivery has sometimes appeared to be the cause, and in other cases there is no apparent explanation of the causation of chronic inflammation which seems to have gone on in the wall of the bowel. Rarely in young persons there is congenital narrowness of the lower part of the rectum. Any hard tumour in the pelvis may press upon the rectum and give rise to stricture. The symptoms of stricture are straining and difficulty in discharging the motions, which consist of numerous small, broken pieces. There is rarely pain in the bowel itself, but this may be felt in the penis, perineum, sacrum, thighs, buttocks, and stomach. Strictures are generally within reach and sight. When not so, the rectum must be explored with vulcanite balls of different size mounted on pewter stems.

Treatment.—When ulceration is present this must be treated as described, but the stricture is also to be dealt with. Slight cases are treated with bougies, the passage of which must never be left to the patient, as deaths have occurred from passing the instrument through the wall of the bowel. Occasionally, the stricture being within two inches of the anus, a short vulcanite tube may be worn at night. Annular strictures near the anus should be divided in a few places with the knife, treatment with tubes following. The incisions go through the induration, reaching, but not dividing, the healthy tissue beneath. Well-oiled wool is inserted for twenty-four hours, and then the tube introduced and worn, except when the bowels act or the rectum is washed out. Strictures are very apt to return. Patients must be warned never to go long without having a bougie passed. In cases of bad ulceration, stricture, and fistula, linear rectotomy is the only treatment of use. The stricture is divided with the knife from its upper edge down to the coccyx, and through its entire depth. A finger is passed through the stricture, and a long, straight knife along the finger. If the incision is kept in the middle line bleeding is trifling only. The wound is to be well syringed and the parts kept perfectly clean. Dry absorbent wool is the best dressing. When incurable, nothing affords relief to these cases except colotomy. For all questions concerning this operation see "Colotomy," vol. ii. p. 187. To sum up the treatment of strictures of the rectum:—

(1) Simple strictures are treated by gradual dilatations with bougies; if they are very resilient, incisions must be made through the strictures.

(2) Strictures complicated by ulceration may be first treated by bougies, but this failing, linear rectotomy is to be employed.

(3) Severe cases continued with fistula and

ulceration require colotomy if linear rectotomy fails to cure.

Half the cases of stricture with ulceration are syphilitic. If tertiary they are incurable. Thoroughly treated with drugs, scraping, etc., in an earlier stage, a favourable result may be procured. The next in order of frequency are the dysenteric, and lastly the tubercular. When the tubercle is local only, good results from inguinal colotomy and treatment of the ulceration. The traumatic cases are generally curable by the means above described.

It is often stated that according to the position and character of ulceration the nature of the disease can be diagnosed. Really, all diseases causing stricture and ulceration may affect any part of the rectum, and the only rules that may be formulated, and they are often broken, are these :—

(1) In syphilitic cases there is ulceration and stricture, a good deal of stricture being present with the ulceration.

(2) In dysenteric cases there is much ulceration, with some tendency to stricture.

(3) In tubercular cases there is much ulceration, rarely with stricture.

(4) In traumatic cases there may be stricture with little or no ulceration. On the other hand, there may be ulceration with no stricture.

Complete excision of stricture of the rectum has been practised. We consider the operation unjustifiable.

Cancer of the Rectum.—The causation of this disease is unknown, and the part which heredity plays is matter of dispute. Middle age is the commonest time of life for its appearance, but the very old may be affected, and we have seen it at thirteen years of age. More men than women are attacked.

Columnar-celled carcinoma is the common and typical form. Sarcomata do occur, and epithelioma is found at the anal margin. Scirrhus, encephaloid, and colloid are the varying conditions of carcinoma. Carcinoma of the rectum is—

(1) Laminar, the wall of the bowel being thickened between its mucous and muscular coats by growths spreading laterally.

(2) Tuberous, the growth projecting into the bowel.

(3) Annular, the growth commencing between the muscular and mucous coats, and spreading round the entire circumference of the rectum.

Cancer of the rectum feels sometimes as though it had a pedicle. This is because it drags upon the mucous membrane. At other times it feels like an ulcer surrounded by nodular, irregular edges, or, high up in the bowel, it may be felt as a hard growth, fixed or movable, combined with ulceration, and more or less completely embracing the circumference of the tube.

Symptoms.—There are some signs which are practically certain indications of cancer. The peculiar odour of the discharge cannot be mistaken by the experienced, and the continuous pain and the feel of the growths are unlike those caused by other rectal disorders. The waxen aspect of face occurs earlier in malignant disease of the rectum than with cancer elsewhere, but in some patients the appearance of vigorous health lasts till the disease is far advanced.

Treatment.—In considering the proper treatment to be adopted for these cases, which generally means settling the question between excision of rectum, colotomy, and no operation at all, the chief points of guidance are—

(1) Age of the patient.

(2) Position and extent of growth.

(3) Nature of symptoms, viz. obstruction of bowels, hæmorrhage, or pain.

If the patient's age is not over forty-five excision is very rarely of advantage. The disease almost always returns in such subjects, even before the wounds are healed, and it is in only very exceptional instances that, on repeatedly removing recurrences, the disease at last ceases to appear. Colotomy prolongs life for a very short period in this class of case, and palliative treatment is generally the proper course. In persons between forty-five and sixty years of age excision is most favourable, and should be attempted when the growth is well within the lower part of the rectum. If it is high, colotomy must be performed, but not until obstructive symptoms are marked and unrelieved by medicine.

For patients above sixty excision is very suitable in cases where there is a fair chance of removing the whole growth. Colotomy is to be employed under the conditions just mentioned. As regards position the growth may be—

(1) Near the anus, healthy mucous membrane being felt above it.

(2) Higher up, but easily felt and moved, although its upper limit is not readily definable.

(3) At the junction with the sigmoid, only just palpable by the finger.

In the first case the growth should be freely excised, in the second Kraske's operation performed, if the patient agrees on understanding its nature and risks, and in the third colotomy is to be done when the symptoms warrant it. We do not believe that rapidity of growth is lessened by a colotomy which diverts fæces from the cancer; many persons by judicious palliative treatment have had life as long prolonged as by an early colotomy. When, however, obstruction is once marked colotomy should not be delayed, and the operation may be needed to check constant spurious diarrhœa, pain, and hæmorrhage. Palliative treatment is employed in cases where excision is out of the question and colotomy not yet required. Rest,

easily digested nourishing diet, alcohol, and sedatives are its chief features. Mild operative measures, as division of the sphincters, or scraping away growth with a scoop, are sometimes of assistance. They are used, of course, only as palliative measures, excision and colotomy being the only effectual methods of removing the pain from cancer of the rectum.

Excision of the Rectum.—The operation is not desirable—

(1) If adhesions exist between the growth and the sacrum, base of bladder, prostate, or cervix uteri.

(2) If enlarged glands are present in inguinal or lumbar regions.

(3) If the patient is already so exhausted by the disease that the necessary loss of blood is a serious risk.

Excision of the rectum is on the whole an operation with very unfavourable prognosis, yet when the patient is seen early enough to make entire removal seem possible, he should be given the chance.

Kraske's method should always be adopted, except when the growth is at the anus, or limited to the neighbourhood of the sphincters; then the perineal operation is advisable. An incision is made in the mid line of the sacrum, beginning at the second vertebra and reaching the anus. The soft parts are divided to the bone and stripped off sacrum and coccyx. The latter is dissected out, and if more room is needed, the lower part of the former cut away with bone forceps. The posterior surface of the rectum is carefully separated, and with the finger its lateral and anterior surfaces also freed. If there is difficulty the bowel should be cut across before it is separated anteriorly. If the disease is low the circumferential cut is made above it, otherwise below it. The division is made with scissors, and vessels are clipped as they are cut. The growth is then cut away and the divided bowel thus dealt with—

(1) The upper end is fixed into the upper angle of the wound, or

(2) An end to end junction of intestinal and anal portions made, or

(3) The upper end is brought down and fixed to the anus.

The first method is usually most suitable. If the peritoneum is opened on removing the growth it is to be closed with silk or catgut sutures.

The perineal operation is only performed when the disease is limited to neighbourhood of sphincters and anus. A deep dorsal incision is the key to the operation. In operating on the male, a silver catheter is to be passed into the bladder and held hooked up under the pubic arch by an assistant. The lithotomy position must be used. On the finger pass a bistoury into the rectum as far as the upper limit of the growth, and make it cut right down on to the sacrum and tip of coccyx. We modify this usual procedure by piercing the skin a little below the anus with a sharp-pointed bistoury, which is made to travel entirely outside the rectum in the cellular tissue up to the top of the growth. Then cut down to the sacrum and press in a sponge to arrest bleeding. Now with a scalpel cut deeply round the anus. Then, with finger in the rectum and thumb in the cut, one blade of a long, blunt-pointed pair of scissors is pushed into the posterior cut, and the other into the cellular tissue of the ischio-rectal fossa. The cellular tissue between the blades is cut through, and the proceeding repeated on the other side. Sponges are put into the incisions on each side, and the outer parts separated from the bowel by broad retractors. We next turn to the perineal part. The rectum is separated by scissors from the tissues around to one inch above the growth. The scissors being kept against the thumb nail, and the finger still inside the bowel, there is no risk of cutting into urethra, bladder, or rectum. The sponges are now taken out, and two large pairs of pressure forceps applied, one to each side of the freed gut. Some short ligatures are tied tightly beyond the clips as these are slowly loosened, and the rectum divided distally. The rectum may be easily removed in this way in ten minutes. A strip of wool is passed along the finger into the bowel, the cavity from which the rectum was removed is stuffed with antiseptic wool, and a T-bandage is tightly applied. In women the assistant's finger should be in the vagina to ensure against the surgeon cutting too near to its mucous membrane. There is little bleeding if the operation is quickly done, and not dividing the bowel when making the dorsal incision ensures greater speed, for the finger in the gut and the thumb outside make rapid cutting safe and easy. The wool is to be removed day by day, that in the bowel being taken out not later than the fourth day. On this day castor oil is given, and the bowels then kept acting every other day. Later, till the parts are healed, a finger or bougie is passed daily. When all is healed a bougie is passed once a week for several months. Whenever it is possible we leave, at the operation, a healthy strip of mucous membrane. Excision of the rectum has sometimes to be combined with colotomy. Sometimes colotomy is needed after excision, because the rectum during healing shows a tendency to troublesome contraction; in other cases the colotomy has been done first to permit of freer subsequent excision.

After complete excision, unless Kraske's method is completed in the second and third ways mentioned, there is incontinence of flatus and fæces. After partial excision there is no more loss of power than after operation for severe fistula. The operation of colotomy is not here described, as full details are given in the author's article on that subject.

Lupoid and Rodent Ulceration of Anus and Rectum.—Lupoid ulcer of the rectum differs from the same malady on the face by being terribly painful and much less curable. The disease is happily uncommon. In its early stages lupoid ulcer resembles a syphilitic sore or a rodent ulcer. It is distinguished from syphilitic and epitheliomatous affections by these peculiarities:—

(1) It does not invade neighbouring organs, contaminate lymphatics, produce hardness, nor form secondary deposits.

(2) It does not lead to stricture of the rectum or any submucous thickening. Its shape is irregular, neither edge nor base is at all hard, and the mucous membrane around is abruptly healthy. It destroys completely as far as it extends, and it tends to spread superficially, and upon mucous membrane rather than upon skin. It often remains stationary, or repair may even proceed rapidly only to be suddenly and rapidly replaced by the original condition. The subjects of lupoid ulcer are almost always tuberculous, and phthisis is generally the cause of death. Tubercle bacilli have been found in the ulcer. The sore is to be thoroughly scraped away with Volkmann's spoon, but benefit is only temporary. Colotomy has been followed by great improvement in the condition of the ulcer.

Rodent Ulcer.—Sufferers from this complaint are rarely under fifty years of age, and usually look ruddy and well. The ulcer resembles that last described, except for its hard, well-defined edges and hard base. The surface is generally red and dry. Free excision is the only useful treatment.

Villous Tumour of the Rectum.—This is a rare disease. The growth is just similar to that of the same name found in the bladder. It is attached to the bowel by a broad base, and the peduncle may be as long as three inches. Usually the appearance of having a pedicle is due to the tumour weighing down the mucous membrane. Generally the tumour springs from the posterior wall. The symptoms are:—

(1) Descent of the tumour on the bowels acting, or even merely on walking.

(2) Discharge of glairy mucus.

(3) Hæmorrhage.

The patient is usually of advanced years. In some cases epithelioma has replaced the removed growth. The growths are sometimes spontaneously shed. They may attain to a large size.

Coccygeal Pain.—Severe pain in female patients is sometimes due to tubercular or rheumatoid arthritic disease of the sacro-coccygeal joint, or to osteitis of the coccyx itself. In other cases it is owing to adhesion in the joint following injury. The cases are intractable, and sometimes only cured by removal of the coccyx.

Proctitis occurs in a chronic and an acute form. The former appears in old people, with the symptoms of heat and fulness in the rectum, frequent desire to go to stool, and great tenesmus; there may be, too, discharge of blood and mucus. Sedative and astringent injections give relief.

Acute inflammation of the rectum resembles dysentery, but lacks the abdominal pain and tenderness and the constitutional disturbance. The bladder is affected, and there is difficulty in passing water. Leeches around the anus, hot baths, and injections of small amounts of water as hot as can be borne are effective treatment. No purges should be given. Light, unstimulating diet and the recumbent posture are to be instituted.

Rare Growths.—Dermoid cysts, angiomata, and lipomata of the rectum are surgical curiosities of such rarity that they do not here demand attention.

Rectus.—Straight, especially in the terminology of the muscular system; *e.g.* the *rectus abdominis*, *rectus capitis anticus* and *posticus*, and the *rectus femoris*. *See* Brain, Affections of Blood-Vessels (*Paralysis*, *Rectus Clonus*); Muscles, Traumatic Affections of (*Rupture of Rectus Abdominis*, *Cruris*, etc.).

Recurrence.—The return of a state or event, *e.g.* of a high temperature (*e.g.* recurrent or relapsing fever); in a special sense, recurrence may mean the reappearance of traits long absent, *e.g.* atavism. *See* Hernia (*Operative Treatment*, *Recurrence*); Larynx, Neuroses of (*Innervation of the Larynx*, *Recurrent Laryngeal Nerve*); Tropics, Unclassed Fevers of (*Recurrent Fevers*); Undulant Fever (*Synonyms*).

Recurrent.—Returning, *e.g.* the recurrent laryngeal nerve or relapsing fever.

Red Blindness. *See* Colour Vision (*Red Blindness*).

Red Corpuscles. *See* Blood (*Corpuscular Elements*); Chlorosis (*Pathology*); Erythrocytes; Physiology, Nutrition (*Blood and Lymph*, *Cells of Blood*).

Red Gum.—Eucalyptus gum (*see* Eucalyptus Gum); also strophulus, or a red papular eruption seen in infants.

Red Lead.—Red oxide of lead or minium, which may be a cause of danger to health. *See* Trades, Dangerous (*Lead-Poisoning*).

Red Lotion.—Lotio rubra. *See* Lotio Rubra; Zinc.

Red Marrow. *See* Physiology, Blood and Lymph (*Source of the Erythrocytes*).

Red Sanders-Wood. *See* PTEROCARPI LIGNUM.

Redlands. *See* THERAPEUTICS, HEALTH RESORTS (*American*).

Redressement Forcé.—The forcible correction of a deformity.

Reducing.—Depriving of its oxygen, *e.g.* a substance ("reducing substance") occasionally found in the urine in tuberculous meningitis (*q.v.*).

Reduction. *See* HERNIA (*Treatment, Reduction, False, "In Mass,"* etc.).

Reduplication. *See* HEART, AFFECTIONS OF MYOCARDIUM AND ENDOCARDIUM (*Physical Examination, Auscultation*).

Redux Crepitus.—Small mucous râles heard in auscultation at the beginning of the stage of resolution (*redux*, a return to health) in pneumonia. *See* PNEUMONIA.

Red-Water Fever.—Texas Fever. *See* PARASITES (*Sporozoa of Texas Fever*).

Reflex and Reflexes. *See* SPINAL CORD, MEDICAL (*Symptomatology, Reflexes*); TENDON-JERKS. *See also* ALCOHOLISM (*Chronic, Impairment of Reflexes*); ANGINA PECTORIS (*Varieties, Reflex Angina*); ASPHYXIA (*Phenomena, Abolition of Reflexes*); BRAIN, PHYSIOLOGY OF (*Scheme of Light Reflex, Fig.* 9); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Paralysis, State of Reflexes*); BRAIN, TUMOURS OF (*Localising Symptoms, Reflexes*); BRAIN, SURGERY OF (*Concussion and Compression, Symptoms*); DIARRHŒA (*Varieties, Reflex*); EPILEPSY (*Etiology, Reflex*); HEMIPLEGIA (*Motor Symptoms, Reflexes*); HYDROCEPHALUS (*Chronic, Symptoms, Reflexes*); HYSTERIA (*Motor Disorders, Reflexes*); NERVES, NEURALGIA (*Etiology, Symptoms*); NOSE, NASAL NEUROSES; PARALYSIS (*Spastic Paralysis, Landry's Paralysis, Acute Anterior Poliomyelitis, Progressive Muscular Atrophy*, etc.); PHYSIOLOGY, NEURO-MUSCULAR MECHANISM (*Reflex Action*); PHYSIOLOGY, SPINAL CORD (*Reflex Functions*); PUPIL (*Size*); SPINAL CORD, MEDICAL (*Physiology, Symptomatology, Reflexes*); TABES DORSALIS (*Symptomatology, Reflexes*); TETANY (*Symptoms, Reflexes*).

Refraction.

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See also GLAUCOMA (*Symptoms, Changes of Refraction*); RETINOSCOPY; etc.

REFRACTION is the name given to the change of direction from their former course of what are conventionally termed[1] "rays of light" when they pass obliquely from one medium into another of different density. It is accounted for by the altered velocity of light in the second medium. In water, for example, light travels at three-fourths, and in glass at two-thirds, of its speed in air. The "optical density" of various substances, by which we mean their power of retarding light, varies pretty closely with their physical densities, as if retardation of light depended on the quantity of matter contained in a given space. When light falls on a plane surface bounding two media, as, *e.g.*, when it passes out of air into plane glass, the perpendicular ray (O*a* in Fig. 1) is undeflected, but all other rays fall obliquely, and are deflected towards the perpendicular ray, as if they came from a point O′ farther away from the glass. This point O′ is called the *virtual image* of O, because to an eye situated in the substance of the glass, as illustrated in the figure, O would appear to be at O′.

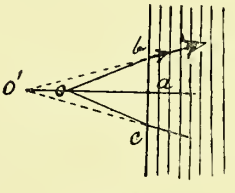


FIG. 1.

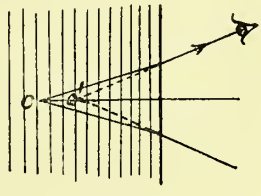


FIG. 2.

The distance O*a* is to the distance O′*a* as the speed of light in glass is to its speed in air, namely, as two to three. To find the expression for the optical density of a substance we have to invert this fraction, for the speed of light varies inversely with the density of the substance through which it passes. The fraction thus inverted is called the "index of refraction" for that substance, and is represented by the letter μ. In the case of glass μ is but slightly over 1·5.[2]

When light travels out of a denser into a rarer medium, as out of glass into air (Fig. 2), the

[1] Rays of light are merely *directions* in which light travels, being perpendiculars to the wave-fronts, and have no concrete existence, but this is not the place to enter into physical optics.

[2] The term density-constant might be suggested as a better one than "index of refraction," if Sylvanus Thompson's terms of *velocity-constant* be adopted for the retardation of light, and since one of these constants is always the reciprocal of the other for any given substance, there is peculiar appropriateness in his selection of the letter h for the velocity-constant, for h inverted closely resembles μ.

obliquely incident rays are bent away from the perpendicular, as if they came from a point called the "virtual focus," whose distance from the surface is two-thirds that of the real focus.

To an eye in air, therefore, as illustrated in the diagram, the point O appears to be at O′.

Plane Glass.—When light passes through a sheet of plane glass the refractions at the two surfaces, being respectively incident and emergent, and thus of opposite kinds, neutralise each other, so that the rays emerge parallel to their previous course before incidence.

Prisms.—When two plane faces of a transparent body are inclined to one another they enclose an "optical prism." A ray of light (*i* in Fig. 3) falling on one face is deflected towards the normal (*n*), and on emerging from the second face is deflected from the normal (*n′*), so that

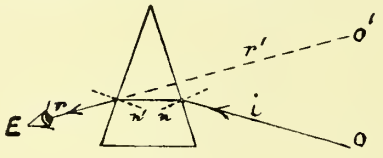


FIG. 3.

the angle of total deflection lies between the incident ray (*i*) and the emergent ray produced backwards (*r′*). It will be seen from the figure that prisms deflect light towards their bases, and appear to displace objects towards their apices. Thus to an eye at E, the object O appears displaced to O′ by an angle which is called the *angle of deviation*.

When the material of the prism is glass, and provided also that the prism be a fairly weak one, and be so held that the light does not traverse it too obliquely, the angle of deviation is about half the angle between the two faces. A prism deflects light least (or, in other words, is held in the "position of minimum deviation") when the incident and emergent rays are symmetrically inclined to its surface. The actual angle between the faces was that by which prisms used to be marked, and frequently are still. A suggestion[1] to measure prisms by the deviating angle instead of the material angle was published in 1886, since it is their optical properties, and not their physical conformations, that make them of value, and an easy method of so doing was placed by the writer in the hands of Messrs. Curry and Paxton. Jackson, independently and more publicly, proposed at a later date that all prisms should be *numerated* by their deviating angles. To distinguish this notation from the old one, it was agreed by a committee of the U.S.A. Ophthalmic Society that the letter *d* should be appended. Thus a prism of 3° *d* is the same as a 6° prism of the old marking; though it deflects light by 3°, the angle between its faces is 6°.

[1] Syme Surgical Fellowship Essay, Edinburgh University (1884), published 1886.

Lenses differ from prisms in that they have curved surfaces. They are divided into two classes, *spherical* and *cylindrical*. The former may be bi-convex (*a*, Fig. 4), bi-concave (*b*), plano-convex (*c*), plano-concave (*d*), convex menisci (*e*), or concave menisci (*f*). Every lens has an unchangeable optical strength peculiar to itself, and corresponding therewith a definite focal length, these two quantities being reciprocally related to each other, for the shorter the focal length the greater the optical strength. It is necessary, therefore, to explain what is meant by the focal length. A convex lens interposed in the path of a parallel beam of light brings the light to a focus at a constant distance from itself, which is called the "focal length of the lens" (Fig. 5). The stronger the lens the shorter its focal length, so that if we let F stand for the optical strength, and *f* for the focal length, $F = \frac{1}{f}$. Ophthalmologists used formerly to measure *f* in inches, but now in metres, since the accepted unit of optical strength is that possessed by a lens of one metre focal length. This accepted unit of optical power is called a *dioptre*. Two lenses each possessed of one dioptre of refractive power placed in close apposition would form a combination having twice the refractive power of either one alone; hence the focal length of the combination would be half as much, *i.e.* half a metre. Three such apposed lenses would have an optical power three times as great, and therefore a focal length three times as short or one-third of a metre; and so on. In fact we have only to imagine ourselves possessed of a metre rod with no markings on it, and nothing else to measure with, to understand the dioptric system. This rod will be about 40 inches long. How many dioptres has any given lens? Just as many as the times its focal length is measurable into the metre rod, or, in other words, as many as the times its focal length in inches will go into 40 inches. Take, *e.g.*, a lens of 10 inches focal length; it will measure four times into the rod, and therefore be of four dioptres. This lens is written down as +4D, the plus sign being allotted to convex lenses. To concave lenses, on the other hand, the minus sign is prefixed. The latter have no *real* focus,

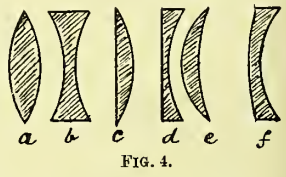


FIG. 4.

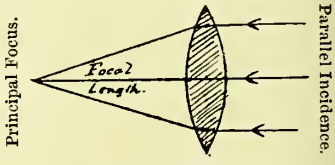


FIG. 5.—A collecting lens: focal length *positive*.

but only a *virtual* one, for when placed in a beam of light they make it spread out as if coming from the virtual focus P′ (Fig. 6). It will be seen that the virtual focus of a concave

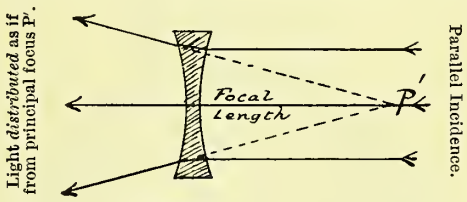


FIG. 6.—A distributing lens: focal length *negative*.

lens lies on the incident side of the lens. It is for this reason the focal length (f) is reckoned negative.

Conjugate Foci.—When divergent rays, such as proceed from a source at a finite distance, fall upon a convex lens, part of the converging power of the lens is taken up in overcoming the divergency of the incident light, leaving only the remainder to converge the emergent light. But lessened convergence of the emergent light means greater distance of its focus from the lens. Hence, though the lens is just as strong as the lens in Fig. 5, the focus lies farther from the lens than the principal focus in Fig. 5. In such case the focus of origin and the focus of destination are called "conjugate foci," because they are so yoked together that when one is made to approach the lens the other recedes from it. The reason of this is very simple,—the more the point of origin approaches the lens the greater is the divergence of the incident light, and the greater proportion, therefore, of the power of the lens is used up to overcome that divergency, leaving only the residuum of power to effect the convergence of the emergent light.

To give an example, let us suppose the lens in question to possess four units (dioptres) of converging power. That means that it converges parallel light to a focus a quarter of a metre away from itself. Let us replace the incident beam of parallel light by one of divergent light from a point half a metre away. Such incident light arrives at the lens with a divergency of two dioptres. It therefore appropriates two dioptres of power from the lens to overcome its incident divergency, leaving two others to effect the convergency of the emergent light. Since the power of the lens is in this instance divided into two equal parts, it is evident that the two foci (of origin and destination) will be equidistant from the lens, and that each will lie twice as far from it as the principal focus. Had the lens been one of 5D instead of 4D, it would retain 3D of converging power, since 2D from 5D leaves 3D, and the conjugate focus of the emergent light would be found one-third of a metre away from the lens. This is illustrated in the figure.

If, on the other hand, the incident light be made to fall in an already converging condition upon the same lens, the whole power of the lens is available to *add* to the already existing convergency. Thus light with 3D of convergency, incident on a +5D lens, would, *but* for the interposition of the lens, have been collected to a focus a third of a metre farther on. When a lens is interposed, however, though the incident light is still directed towards that focus, it becomes a virtual focus, since the light does not really arrive there, being gathered up by the lens to a real focus at a nearer point. If the lens be one of +5D, it adds the whole of its 5D to the 3D already possessed by the incident beam, so the light leaves it with a convergency of 8D. The conjugate focus, therefore, will be $\frac{1}{8}$ metre from the lens, for we still call them conjugate foci, though one is virtual and the other real. They both lie on the same side of the lens, and the nearer one is brought to the lens the nearer comes the other. It has been observed that conjugate foci, of whatever kind, always move in the same direction.

Images.—So far we have supposed the incident light to proceed from a point. But in nature luminous points do not exist except in groups. The surface of any object may be regarded as composed of an indefinite number of points side by side; and since, when a lens is brought into play, each has its conjugate focus, or point-image, all of these also lie side by side, so as to form a corresponding group, or *image* of the object, which may, however, be real or virtual, just as we have already considered. The relative size of image and object follows the very simple rule that it is directly proportionate to the distance of each from the lens.

When an image lies on the opposite side of a lens from the object, it is always *inverted*; when on the same side it is always erect.

Cylindrical lenses differ from spherical in that they are curved in one direction only, but like them they are divided into two classes of convex and concave. The shape of a convex cylinder is as though a small circular piece were sliced off the surface of a column or solid cylinder of glass. One meridian of the lens, therefore, answering to the long axis of the column, has no refracting power, and is called the "axis" of the lens. The meridian at right angles to the axis has the maximum refracting power, and is that by which the cylinder is numerated. Thus a cylinder having its maximum curvature equal to the all-round curvature of a +1D spherical lens is a +1D Cyl. Its focal length is 1 metre, but the light is focussed into a line instead of into a point.

The extremities of the axis are usually denoted by tiny scratches in the glass, but they might with advantage be further indicated by two notches in the metal rim of the lens, so as to utilise the surgeon's sense of touch, according

to a suggestion made to the writer by Anderson Stuart. It is customary also for good cylindrical lenses to have two opposite areas obscured like "ground glass," having their edges parallel to the axis of the lens.

Measurement of Lenses.—Concave lenses not only have the property of making objects viewed through them appear smaller, but when the lens is moved from side to side an apparent motion is imparted to any object viewed through the lens in the same direction as the movement of the lens (*see* Fig. 7). The farther the lens is held from the eye the greater is the apparent motion. The strength of the lens is best determined by the process of "neutralisation," *i.e.* by finding which of the convex lenses from the trial-case abolishes this apparent motion and

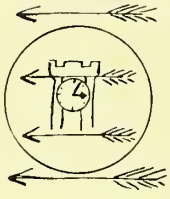


FIG. 7.—Apparent motion through a concave lens.

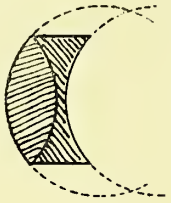


FIG. 8.

makes the combination resemble a piece of plane glass. For weak lenses this method is very exact, and for strong lenses it is exact enough for practical purposes. When, however, strong lenses of equal power and opposite sign are apposed, the combination exhibits an appreciable "convex" character of motion, whichever lens be held nearer the eye. This fact, which many have noticed from the trial-case and puzzled over, I have graphically explained by Fig. 8, which shows how the two outside surfaces of the combination would, if continued in space, as shown by the dotted lines, form a convex meniscus.

A convex lens not only makes distant objects appear dim to an emmetropic eye and near objects appear magnified, but when displaced laterally it makes objects appear to move in the opposite direction, *i.e.* *against* its own motion (Fig. 9). This motion can be neutralised, and the lens measured by the apposition of the concave lens of equal strength from the trial-case. An excellent alternative mode of measuring lenses is afforded by the now well-known Geneva lens measure (Fig. 10), which, when pressed against each surface of the lens in turn, tells the dioptric power of each surface. The two readings must, of course, be added together to give the strength of the lens. For pebble lenses the findings of this instrument are a little too low. The index of refraction being higher for pebble, the same curvature betokens a greater refractive power.

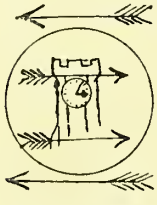


FIG. 9.—Apparent motion through a convex lens.



FIG. 10.—Geneva Lens Measure.

Measurement of Cylinders.—The first step is to find the axis, by looking through the lens at a (preferably distant) vertical line and rotating it till the part of the line viewed through the cylinder no longer appears "tilted," but continuous, above and below, with the rest of the line. If now the lens can be moved in a direction parallel to the length of the line, without causing any apparent displacement of neighbouring objects, the axis of the lens is parallel to the line. If, on the other hand, it imparts apparent motion to the neighbouring objects when it is moved along the direction of the line, the axis is perpendicular to the line, as confirmed by the apparent immobility of objects when the lens is moved at right angles to the same. In either case the lens is convex or concave, according as the objects appear to move "against" or "with" its own motion. Having found the axis and character of the lens, it is easy to appose successive cylinders of opposite sign from the trial-case, axis to axis ever, till all apparent motion is neutralised. Another plan is to appose spherical lenses till one is found which annuls all motion along a meridian perpendicular to the axis, since this is the meridian of maximum curvature.

References have already been made to the Geneva lens measure. If pressed against a cylindrical lens, its index-hand points to zero when its three teeth lie along the axis of the lens, but on rotating the apparatus slowly the index rises gradually till the maximum reading is reached at right angles to the axis, demonstrating very prettily the gradual increase of curvature of successive meridians as they become more and more distant from the axis. In the case of strongly cylindrical spectacle-lenses, the exact inclination of the axis is a matter of considerable importance, and though

it may be guessed to a very close approximation by the methods already described, greater accuracy is afforded by the writer's "axis-finder," which tells the axis not merely in relation to the long diameter of the lens, but to the long diameter of the spectacle frame as a whole, as it sits upon the face, which is, of course, what we want to know (*see* Fig. 11).

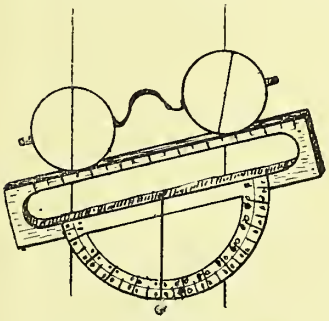


FIG. 11.—Writer's Axis-Finder.

EMMETROPIA is the optical condition of the natural eye, in which, during negative accommodation, the length of the eyeball and the focal length of the dioptric media are so proportioned that the retina lies at the principal focus; or, in other words, the conjugate focus of the retina lies at infinity. This enables the retina to receive clear images of very distant objects, and, when the ciliary muscle is paralysed, of them only. With an active ciliary muscle each increment of accommodation causes the conjugate focus of the retina to approach the eye, till eventually it reaches the "near point" or "punctum proximum."

All investigators are agreed that accommodation is brought about by a change in the shape of the crystalline lens effected by contraction of the ciliary muscle, though two rival theories (Helmholtz, Tscherning) are in the field as to the nature of the change, the former having maintained that it is caused by relaxation, and the latter by tightening, of the zonule. It is readily seen that while an emmetropic eye has the power of accommodating itself to receive the diverging rays which proceed from near objects, the utmost it can do by extreme relaxation is to focus parallel rays on its retina. The smallest convergence possessed by an entering pencil is incompatible with distinct retinal pictures. That is why an even weak convex (*i.e.* converging) lens held before an emmetropic eye impairs the definition of distant objects, a phenomenon which supplies us with one of the three tests necessary to prove an eye emmetropic. Since it only excludes hypermetropia, a second test is needed to exclude myopia, and consists in demonstrating that either there exists full acuity of distant vision, or that at any rate there is no marked improvement of vision (other than accounted for by slight contraction of the pupil) by a weak concave lens. Though the second test excludes high astigmatism, yet a third is necessary to guard against the presence of a low degree of that defect, and may be instituted either by noting the effect of weak cylindrical lenses or by directing the patient's attention to lines running in different directions, as described farther on.

Actually, very few eyes are strictly emmetropic, a trace of astigmatism being almost universal, owing to the vertical meridian of the cornea being more strongly curved than the horizontal. While any distant object may be chosen for the first of the two tests, the most convenient in practice are Snellen's test-types (consult articles on "Visual Acuity") at a distance of 6 metres (20 feet).

Range of Accommodation.—In emmetropia the "far point" remains at infinity, but the "near point" gradually recedes from the eye as age advances, curtailing the range of accommodation year by year. The near point is best discovered by approaching Jaeger's small test-types (described in the article on "Visual Acuity") first to both eyes together (binocular), then to each singly (monocular), till the greatest nearness compatible with distinct vision is reached. This distance, measured from the eye in centimetres and divided into 100, gives the range of accommodation in dioptres. Thus a patient who ceased to see J_1 (see article on "Visual Acuity") quite distinctly if held nearer than 8 cm. to his eyes, has $\frac{100}{8} = 12\frac{1}{2}$D of accommodation power, while another, whose power of distinct vision fails at 25 cm., has $\frac{100}{25} = 4$D of accommodation power. Dioptric tapes are sold which make even this simple calculation unnecessary. Those who prefer to take measurements in inches should divide into 40 instead of into 100, and the result will come out the same. Thus, with a near point of 5 inches, the range of accommodation is $\frac{40}{5} = 8$D. In later life a normal eye acquires slight hypermetropia.

The following table shows the range of accommodation at different ages, and is taken principally from Donders. The acquired hypermetropia appears to the writer slightly exaggerated:—

Age.	Near Point in Inches.	Near Point in Centimetres.	Range of Accommodation.	Acquired Hypermetropia.
	"	cm.	D.	D.
10	$2\frac{3}{4}$	7	14	
15	$3\frac{1}{4}$	8	12	
20	4	10	10	
25	$4\frac{3}{4}$	12	8·5	
30	$5\frac{3}{4}$	14	7	
35	$7\frac{1}{4}$	18	5·5	
40	9	22	4·5	
45	$11\frac{1}{2}$	28	3·5	
50	16	40	2·5	
55	26	66	1·75	0·25
60	80	200	1	0·5
65	∞	∞	0·75	0·75
70	−40	−100[1]	0·25	1·25
75	−23	−57	0	1·75
80	−16	−40	0	2·5

[1] The minus sign is prefixed to show that the near point has become virtual, *i.e.* behind the eye instead of before it.

Hypermetropia is generally the expression of imperfect development of the eyeball, which resembles those of the lower animals in being more flattened from before backwards. Thus results a discrepancy between the length of the eyeball and the focal length of its media, the former being less than the latter. When the accommodating power of the eye is able to shorten the focal length sufficiently to make it coincide with the eyeball length, the defect of focus is for the time overcome. Hypermetropia of this kind is called "Facultative," in contrast to "Absolute" hypermetropia, in which all such efforts fall short of their mark. Moreover, all young hypermetropes, and many middle-aged ones, have a certain fraction of their defect (which becomes smaller every year) constantly masked by an involuntary residuum of accommodative action which remains in force even when the voluntary relaxation of accommodation is tempted to the utmost by convex lenses. This perpetually neutralised fraction of the refractive error is called the "Latent H," while the remainder is called the "Manifest H," since it can be manifested by the toleration of convex lenses. The two together constitute the "Total H." To learn the "Latent H," we must first measure the "Manifest H," then paralyse the ciliary muscle by some cycloplegic like atropine, and measure the "Total H." The difference between the two gives the "Latent H." To detect H we may employ retinoscopy (*q.v.*), or subjective tests. For the latter it is customary to place the patient 5 or 6 metres from Snellen's distant test-types, and find, with each eye separately, what is the *strongest* convex lens compatible with the best vision obtainable. If, after thus testing each eye separately, the two lenses found be left in the trial frame, while, with both eyes open, weak pairs of sphericals are placed simultaneously before the two eyes, it will often be found that more hypermetropia becomes manifest binocularly than monocularly, and this procedure should never be omitted in correcting any error of refraction. Before turning to the treatment of H, it is well to consider that nature treats it by hypertrophy of the ciliary muscle (analogous to compensatory hypertrophy of the heart). So long as compensation is sufficient and no discomfort exists, it is wise to let nature develop her own resources, and to refrain from what we might almost call meddlesome spectacling. It is chiefly in strong, healthy young people that such compensation exists. There is, however, a larger class in these hyper-educational days whose neuro-muscular energy is insufficient, and in whom the lack of compensatory power makes itself felt by headaches, eye-strain, and various sensory discomforts, while at times the page becomes misty, as the tired muscles "give out." Even in such cases we should not always think of correcting the total hypermetropia. We should help nature rather than supplant her. In some cases, especially among young people, it suffices to correct the manifest hypermetropia alone; in a larger number half the latent element should also be corrected, while in a certain proportion, and I think in all adults, and most certainly in every case of convergent squint, it is advisable to correct the whole. We should also be guided often by the muscular balance of the eyeballs.

If, while looking at a distant object, a condition of latent convergence can be demonstrated, a more complete correction of the total hypermetropia is indicated.

If, on the other hand, latent divergence be great, convex glasses would increase it by lessening the accommodative effort, and with it the associated convergence, and should therefore be prescribed as weak as possible. The balance is conveniently tested by placing a lighted candle, or other small source of light, at a distance of 5 metres from the patient, while a disc of horizontal glass rods [1] is held before one eye so as to make the flame appear drawn out into a long streak of light, while the naked eye, of course, sees the flame as it is. Homonymous diplopia, in which the streak appears on the same side as the rods producing it, indicates convergence, while crossed diplopia indicates divergence.

The slight hypermetropia incidental to diabetes is attributed, with good reason, to an increased index of refraction of the vitreous body. Increased index of the aqueous, on the other hand, tends to myopia. Nearly all young children are hypermetropic, and there is reason to believe that it gradually lessens with development of the eye. After about the age of fifty-three, however, another kind of H (H acquisita) begins to appear, and goes on increasing, as shown in the last column of the table given in the section on emmetropia. Priestley Smith ascribes it, with some reason, to the steady growth of the lens which he has so beautifully demonstrated, but it is also rational to expect that the eyeball should share the senile shrinking of the whole body, and thus its antero-posterior diameter becomes less (Donders).

Since in hypermetropes part of the accommodative power is spent in correcting the refractive defect, the range of accommodation is shortened in proportion, and the near point lies farther from the eye. A natural consequence of this is that presbyopia sets in earlier.

Hypermetropic patients are apt to have the curious habit of finding relief by pressing the inner canthi with their forefingers. It only remains to add that ocular headaches, hyperæmia of the lids, and sometimes even blepharitis, are kept up by hypermetropia.

[1] The disc of rods is obtainable from Messrs. Curry and Paxton, 195 Gt. Portland Street, together with scales for placing behind the flame in order to measure the deviation in degrees.

Myopia exists whenever the antero-posterior axis of the eyeball is too long for the focal length of the eye. Pencils of light must enter the eye divergent if they are to be focussed on the retina of a myopic eye. Compare what is said about Fig. 7. There are, optically considered, four kinds of myopia—

(*a*) From abnormal suspension of the lens.
(*b*) From densification of nucleus.
(*c*) From the shape of the cornea.
(*d*) From elongation of the globe.

The first is well known as a transitory consequence of a blow on the eye, and is then termed traumatic myopia. Probably the suspensory ligament is stretched by the blow, and allows the lens to come forward a little until its elasticity is re-established. It may also allow the anterior surface of the lens to assume an abnormal rotundity.

Possibly a similar myopia may exist from defective development of the zonule apart from traumatism.

The second is frequently met with up to 3 or 4 D, or sometimes much higher, in the early stages of nuclear cataract.

The third is the least considerable class, and is discovered when the ophthalmometer shows the cornea to be strongly curved. Care should be taken not to overlook a low degree of conical cornea.

The fourth kind of myopia is by far the commonest. In low degree it sometimes occurs in a purely developmental form, the eye being simply larger than usual in all its dimensions. Happily in these cases the liquid pressure within the eye (which is generally about that of a inch of mercury) is less than usual. Were it not so the short sight would be almost certain to increase, in virtue of the simple hydrostatic law, to be remembered in this connection, that for a given pressure per unit area the strain on the coats is proportionate to the number of unit areas exposed to the pressure. It is very likely that some of the myopias which are assumed to be developmental because of the uniform enlargement of the eye are also really stretched eyes, for a study of hydrophthalmos and infantile glaucoma shows that the eyes of children are more distensible in all diameters, whereas those of (at any rate myopically predisposed) adults are more distensible at the posterior pole. One reason for this is, that the more careful and prolonged inspection of near objects, which comes with growing years, requires greater action of the extra-ocular muscles, which support the globe everywhere except at the posterior pole, where the muscular cone leaves it to be supported only by retrobulbar fat.

Unlike hypermetropia, myopia is the rarest thing in infancy, and generally does not show itself till between eight and twelve years of age. The educational period of life from twelve to eighteen is the most dangerous for its increase, since it requires prolonged convergence of the eyes upon near objects, with more or less stooping, and often in an atmosphere which is not favourable to the vital resisting power of a fibrous structure like the sclera.

Cohn has shown that short sight is rarer in the country than in towns, in elementary schools than in advanced schools, in both than in the universities, in the lower forms of a school than in the higher, and in the lower classes of society than in the higher.

The indications are obvious:—

(1) To correct the refractive error, in order to prevent stooping and to lessen the convergence.

(2) To see that school-rooms are well lighted and ventilated.

(3) To have desks and seats adjustable to the height of each several scholar, according to well-defined rules.

(4) To use books well printed on unglazed paper, and to teach bold, upright writing.

(5) To discourage long evening work, and bookworm habits out of school hours, and to forbid impositions.

(6) To start, if possible, special schools, or separate departments in schools, for short-sighted children, with modes of teaching adapted to give a useful education with the minimum strain on the eyes. It is hardly fair to expect the methods of an ordinary school to be altered for the benefit of a small minority, or to allow unsound eyes to compete with sound ones.[1]

(7) To exchange sedentary habits for fresh air and exercise, and to guard against inactivity of the liver and primæ viæ, which have a deleterious effect on short sight.

(8) During the attacks of choroidal congestion, to give up near work, and advise suitable medical treatment.

(9) To avoid working the eyes under conditions of bodily fatigue, or with insufficient food.

Returning to the first of these indications, there is no doubt that, except in higher pathological cases (of which more anon), the full optical correction constantly worn is the best treatment in the case of young myopes, and affords the greatest security against much progress of the affection.

An exception to this has to be made in the case of children whose calling in life would be seriously compromised by the wearing of glasses; army candidates, for instance, may be advised to abjure glasses altogether until medical examination is over, since their use undoubtedly leads to forgetfulness of that ingenious dexterity of unravelling the details of hazy objects acquired by nearly all uncorrected myopes.

[1] An attempt in this direction has been made near Oxford by Mr. Doyne, and is being made in Bournemouth by the writer.

Donders was the first to point out that though glasses for distant vision are a luxury, it is for near vision they should be considered a necessity, however much the child may prefer to be without them. If, however, we slightly under-correct children, who love to see objects clearly, we shall, as Jackson observes, tempt them to look obliquely through their glasses in order to see better, and thus by straining their muscles and introducing the astigmatic effect of oblique lenses, derive more harm than good from their spectacles. To make sure of not exceeding the full correction in myopes who possess an active accommodation is by no means easy, and a mydriatic is more often necessary in myopia than in hypermetropia, except when we decide to under-correct, as we may do in people who will not misuse their glasses. Since by the exercise of accommodation objects can be seen with equal distinctness through lenses which over-correct the myopia, great care should be taken to select the *weakest* glass that gives full distant vision.

This rule, it will be remembered, is the opposite of that for presbyopia, where the *strongest* convex glass had to be chosen.

When glasses are taken to for the first time by adults, it is generally needful to prescribe a reading pair weaker, by 1D or more, than the distant pair. In presbyopia combined with myopia the reading-glass should be weakened by the amount of presbyopia.

There are cases of congested choroid and irritable retina in which, during the greater part of the day, it is inadvisable to wear glasses at all, since myopic haze gives the retina more rest than the continual appeal of well-seen objects. Such cases are liable to periodical attacks of congestion of the eyes, due not only to their over-use, but also to a constitutional disturbance not yet fully understood, and it is particularly at these times that the myopia increases and the greatest dangers are run of choroiditis or hæmorrhage at the macula. It is to aggravated cases of this kind that the name malignant myopia is applied. The ophthalmoscopic appearances are described in their own section of this work.

In prescribing glasses for high myopia two or three precautions are necessary. The first is to remember that the spectacle lenses will be worn nearer the eye than the lenses in the trial frame, and thus will over-correct the defect if the trial lenses exactly correct it. A glance at Fig. 6 will show that a lens exactly corrects a myopic eye when its principal focus coincides with the far point. When, however, the same lens is brought nearer the eye, its principal focus lies within the far point, so that the light enters the eye with a greater divergency than is required to correct the myopia.

This excess of divergency, though overcome by the patient's accommodation, induces eye-strain all the more for that very reason. A slight deduction, therefore, must be made from the trial lens in proportion to its distance from the eye and also in proportion to its strength. Thus a subtraction of 0·5D may be necessary from a 5D trial lens, of 1D from a lens of 10D, and of 2D from a lens of 20D, or possibly even more. Another precaution is to adjust the centres of strong lenses so as to give the most easy inclination of the visual lines to each other. The stronger a lens the greater is its prismatic effect on the visual line, when this line does not pass through the optical centre. A simple way of solving this problem, in the case of glasses prescribed for distance only, is to place the disc of glass rods in the trial frame, already filled with its lenses, and adjust the breadth of the frame till a distant flame appears to coincide with the streak. The recorded distance between the lens centres may then be prescribed unless it should be at all extraordinary.

Since the range of accommodation is shifted in myopia, by the patient's far point being nearer than infinity as in emmetropia, the near point lies nearer the eye, and its recession does not embarrass the patient till later in life, and in the case of high myopia, never.

Presbyopia.—Presbyopia is the physiological age-element that creeps in with every variety of refraction, and depends on gradual lessening of the range of accommodation from recession of the near point. It is due to decreasing elasticity of the lens, supplemented in later life by atrophy of the ciliary muscle. The condition does not receive the name of presbyopia until the nearest point for comfortable reading begins to recede beyond the distance at which books and papers are generally held. In healthy emmetropia this begins about the age of forty-five, and is evidenced by the necessity of removing near work to a rather greater distance from the eyes than formerly, and later still by the expedient, which the patient discovers for himself, of allowing a quantity of light to enter the eyes while reading in order to contract the pupil and thus increase the definition of the retinal pictures in the same way that a small stop sharpens the definition of an imperfectly focussed camera. The healthy emmetrope would generally require about a +0·5D lens to correct his presbyopia at forty-five years of age, +0·75D at forty-seven, +1D at fifty, +1·5D at fifty-five, 2·25D at sixty, 3D at sixty-five, 3·5D at seventy, and after seventy-five from 4 to 6D, according to the generally more or less diminished acuity of vision from senile changes.

But it is always better to test the patient's near point both binocularly and monocularly, as already described, to make sure that the glasses prescribed make it lie well within the distance at which his calling in life requires his work to be, and which of course differs in different occupations. If the distance of the near point

be measured with a dioptric tape, the range of accommodation can be read off in dioptres, and Landolt's plan for correcting presbyopia is to reckon two-thirds of this range available for continuous work, and add thereto the refractive aid necessary to bring this two-thirds up to the patient's work. For example, if a patient's near point lie $\frac{1}{3}$ metre away, his range of accommodation consists of three dioptres, of which two are available for constant work. Were his work, therefore, to lie at $\frac{1}{2}$ metre habitually, he would not need glasses, but were it to lie at $\frac{1}{3}$ metre, he would need lenses of 1D. Mistakes are more often made by prescribing glasses too strong than too weak, since Donders rather over-estimated the requirements of the different periods of life.

In hypermetropic subjects presbyopia becomes manifested earlier in life, since part of the range of accommodation at its far end is absorbed in neutralising the refractive error, leaving only the remainder available for near work. The corrections specified above for each age in emmetropia should therefore theoretically be added to the lens which corrects the hypermetropia, but actual measurement of the near point to confirm this should not be omitted.

In myopia, presbyopia appears later in life unless glasses are worn constantly, in which case the reading pair should have an abatement of strength from the distant pair equal to the lens that would be required at the same period of life in an emmetrope. It will be seen, therefore, that while a hypermetrope of 1D will require a lens of about 2·25D at fifty years of age, a myope of 1D will scarcely need a glass at all at that age, though at sixty years he will need about +1·5D.

Astigmatism.—Two kinds are recognised clinically — irregular and regular. The former is caused by such defects as corneal nebulæ, corneal facets, unequal refractive indices of the sectors of the lens, and in short irregularities of any unsystematic kind in cornea or lens. It is incapable of correction, except partially by the employment of a stenopæic slit or aperture.

Regular astigmatism is of two kinds, corneal and lenticular, of which the former is by far the most important. The latter is apt to neutralise the former in measure, and it is mostly due to tilting of the lens. Corneal astigmatism exists when the part of the cornea employed for direct vision is spheroidal instead of spherical, or when it is more strongly curved in one meridian than in any other.

The vertical meridian is generally that of maximum, and the horizontal that of minimum, curvature. This astigmatism, therefore, is called "according to the rule." When the reverse is the case the most strongly curved meridian is approximately horizontal, and this condition has received the name of "perverse astigmatism," since it is, with a measure of truth, supposed to give rise to asthenopia more frequently than the other kind, and to require correction more fully. The fact that a low degree of ordinary astigmatism exists physiologically in almost every eye helps to confirm this belief.

Estimation of Astigmatism. — Retinoscopy (*q.v.*) is more serviceable for the detection of astigmatism than for any other error of refraction.

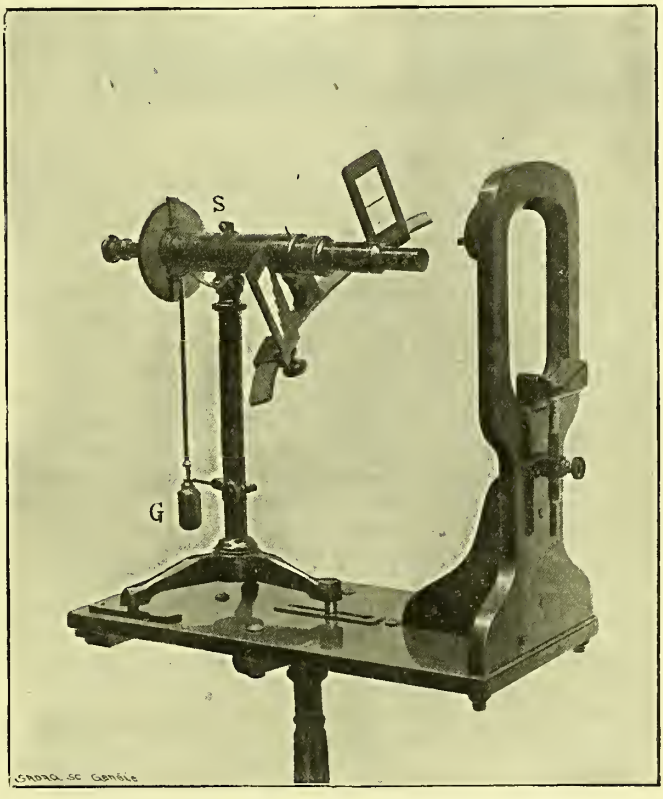


Fig. 12.—Ophthalmometer (as made by Pfister and Streit).

Another valuable objective method is furnished by the ophthalmometer. As arranged by Helmholtz, it was suitable for physiological investigations only, but the later instrument of Javal and Schiotz is more suited for taking clinical observations. It consists essentially of a telescope (Fig. 12), directed towards the patient's eye, and provided internally with a doubling apparatus which produces two images of the cornea looked at. The telescope bears two arms which support white figures on a black ground, one of which is cut out in little steps (Fig. 13). To use the instrument these

figures are placed in a strong light, that the reflections from the cornea may be studied, the arms, to begin with, being horizontal. Since each figure (or mire, as it is called) appears doubled on the patient's cornea, four reflections are seen, of which two lie close together. One mire is then moved until its margin appears to touch that of the other. The curvature of the meridian may now be read off from the instrument, and if after rotating the arms through a quarter of a circle the mires still touch, there is no astigmatism. Should they overlap (Fig. 14),



Fig. 13.



Fig. 14.

it shows the second meridian to be more strongly curved than the first, and to possess as many additional dioptres of refractive power as there are steps in the overlapping. Should, however, the mires, instead of overlapping, have moved farther apart, the second meridian is the less curved of the two, and after bringing the mires together again, the arms should be swung round once more to the first meridian to count the overlapping there. Should one mire appear displaced tangentially with reference to the other, it shows that the meridians of maximum and minimum curvature are oblique, and the arms should be swung round till the tangential displacement is corrected. One of the two required meridians is now found, and the other is at right angles to it.

But after all has been said, the most serviceable tests for intelligent patients are the subjective ones, and all others should be supplemented by it. We may divide subjective tests into type-tests and line-tests. For the former, Snellen's test-types are used, at a distance of 6 metres, while lenses are manipulated before the eye. The best lens to begin with in an eye approximately emmetropic, or after correcting H or M as nearly as may be, is a $-0{\cdot}5$D Cyl., after placing a trial frame on the face with an obturator (*i.e.* an opaque disc) before the other eye. If this lens affects vision equally, with axis placed first horizontal and then vertical, there is no antigmatism, unless of the oblique kind. The next step, therefore, is to place the same lens with axis at 45° on one side of the vertical, and then axis at 135° on the other side. Whenever better vision is obtained with the axis in any one position than in a position perpendicular to it, astigmatism is detected. The lens is now to be rotated till the best position of the axis is obtained, and then left in position while other minus cylindrical lenses of increasing strength are tried in succession before it, with their axes in line with its axis, till the maximum acuity of vision is reached. But during this process care should be taken to try the effect, at intervals, of weak convex sphericals, and if vision is improved, or even made no worse by them, they should be adopted, and be associated with the cylinder in the trial frame. Moreover, the exact inclination of the axis should be confirmed as the examination proceeds, since with increasing strength of cylindrical correction the exact placing of the axis becomes more delicate and easy. Finally, a weak Cyl. of $-0{\cdot}25$D (or a Jackson's lens, which consists of a weak $+$Cyl. combined with a weak $-$Cyl. at right angles to each other) should be placed alternately "axis with" and "axis against" the previously found correction. If these two positions affect vision equally the astigmatism is properly corrected, and the other eye can be proceeded with in like manner.

Under-Correction of Astigmatism.—It is my impression that astigmatism, whether myopic, mixed, or hypermetropic, should be slightly[1] under-corrected, to allow play for the variable element, for though the mass of the astigmatism remains unchanged, there is a kind of fluctuating margin, like the ebb and flow of the tide, dependent on varying tone and contraction of the eyelids. This interesting subject, which has been specially studied by George Bull and George Berry, deserves a longer account than the scope of this article allows.

Line-tests.—Only the best of these need be mentioned, namely, first, those which consist of a group of radiating lines, as exemplified in Snellen's "Sunrise," or the well-known "clock"; and, second, those consisting of one or more groups of parallel lines. A single group can be rotated on the wall, or several groups of lines running in different directions may be placed near each other on a stationary card. A very good example of the last, to which my attention was called by Mr. Lionel Laurence, is made by the "Montreal Optical Company." We may, however, select the "clock" to illustrate the principle of all other line-tests. In this, twelve narrow bands, each consisting of three parallel lines, radiate from a centre. After excluding one eye of the patient, and placing the clock 6 metres off, the weakest minus, or strongest plus, spherical lens should be selected which only just allows one diameter to be distinct. One meridian of the eye has now been made emmetropic, namely, that which is at right

[1] The advice, which the reader may have met with, to choose the weakest minus cylinder, or the strongest plus cylinder, which gives best visual acuity is based on an obvious fallacy. The weakest of either should be chosen.

angles to the brightest ray. All that remains is to appose minus cylinders, with their axes at right angles to the brightest ray, till we find the one which makes rays at right angles to the originally brightest one equally bright also.

It is prudent not to be content with this, but to make a final appeal to the distant test-types, for which it will often be found that a slightly weaker cylinder is best, and I have even met with a case now and then in which a slight difference of axis is preferred. When such discrepancies occur, it appears probable that the line-test is more strictly true, but that the type-tests indicate the lens which it is expedient to prescribe, at least for a time. In testing the second eye, it saves time, in view of the predilection which As. displays for bilateral symmetry, to commence with lenses similar to those already found for the first eye.

Estimation of Refraction. — Though this has been dealt with under each heading, the following general order of procedure may be described :—

Having glanced at the patient's eyes to see whether they are deep set and with contracted pupils, as in emmetropia ; or whether they are prominent and with large pupils, as in myopia ; and whether the eyelids look tired or drawn, and the forehead frowning, as in refractive asthenopia ;—the outer canthus of each eye may be drawn back, while the eye is made to look strongly in the opposite direction to observe whether the side of the globe is short and strongly curved as in hypermetropia, or with a long gradual curve as in myopia. The tension of the eye should be tested as a matter of habit to observe the normal variations. The visual acuity both in distant and near vision (*see* art. on Visual A.) should next be recorded, and the near point measured. Next, one of the two chief objective tests should be applied—retinoscopy (*q.v.*), or ophthalmometry. The latter is described a little farther on. The patient should now be seated at a measured distance of 5 to 6 metres from the distant test-types, with a trial frame on his face bearing the correction already found, and with a circular vulcanite disc, called an obturator, in front of one eye. The best trial frames have obturators hinged on to them so that they cannot be lost. The defect of all trial frames which the writer is acquainted with is that they admit distracting side-lights to the patient's eyes. It would be better were they provided with side screens. The objective finding for each eye should be confirmed by the apposition of weak + and − spherical and cylindrical lenses, and the axis of a cylinder be carefully adjusted, since its position sometimes differs during attention of the eye from that which it occupies when the eye is disengaged, as during retinoscopy. The visual acuity then obtained should be made a note of.

Should no objective test have been made first, the procedure may be as follows :—

A. Covering one eye with the obturator, test the vision of the naked eye by the distant test-types. Should the visual acuity be practically perfect, myopia cannot be present, nor high astigmatism. It remains, therefore, to test for H or low As. To do the first, a very weak and then a stronger convex lens may be put up before the eye. If the legibility of the lower distant types is impaired by a +0·25D, and almost abolished by a +0·75D lens, there is no manifest H. Should the legibility be unaffected by either of these lenses, put up stronger ones till the *highest* power is reached that does not distinctly impair legibility. This lens measures the manifest H (H_m).

B. Should, however, the visual acuity of the naked eye have been poor at first, the case may be one of myopia. Try, therefore, a rising series of concave lenses till the *weakest* is found that affords the best obtainable vision.

C. Next, test for astigmatism, by directing attention to a line-chart with any previously found myopia slightly under-corrected, or hypermetropia over-corrected. If lines in some certain direction stand out more boldly than others, proceed, as already described under "Astigmatism," by putting up concave lenses, with axes perpendicular to these bold lines, till the lines are made equally bright in all directions, and then confirm the result by the test-types. If two consecutive *cylinders* appear to have the same effect, retain the *weakest*, whether + or −. From this remark it will be seen that the rule for the strongest convex and weakest concave does not apply to cylinders, but only to spherical lenses.

To summarise, if the naked eye has

A. Full visual acuity. There is either
   (*a*) Emmetropia, or
   (*b*) Hypermetropia (facultative), or
   (*c*) Low astigmatism.

B. Poor visual acuity. There is either
   (*a*) Myopia, or
   (*b*) High hypermetropia (not fully neutralised by accommodation), or
   (*c*) Astigmatism.

Prisms and the Decentering of Lenses.— It is sometimes desirable to combine a prism with a lens, especially in hyperphoria (*q.v.*) or for diplopia. The simple rule may be made that a prism appears to drive the image seen through it in the direction of its "edge" or "apex." If a simple mnemonic metaphor may be permitted, the image flies before the edge of the sword. If strong, the prism should be divided between the two eyes. The prismatic result can be obtained by decentering a lens, the rule being that for each degree of prismatic effect desired, the lens should be decentered by a distance equal to $1\frac{3}{4}$ cm., divided by the

number of dioptres in the lens. As regards the direction of decentering, since the thinnest part of a concave lens is at its centre, this centre should be displaced in the direction of the thinnest part or apex of the required prism, the reverse being the case with a convex lens, for since its centre is its thickest part it should be displaced in the direction of the base of the required prism. A scale constructed by the writer for the measurement of hyperphoria is obtainable, with directions thereon as to the amount of decentering that is necessary to correct any defect.

It is evidently important in all cases of decentering to know what point to decenter from; in other words, to know the exact distance between the eyes, and whether one eye is higher than the other. Numerous pupillometers exist for this purpose. The most accurate are those that require the patient to look at a distant object through two pin-holes, but this requires too much intelligence for most patients. The pupillometers in ordinary use, which allow the prescriber to take the measurement, are subject to errors from parallax, since they secure no fixed point of view for the examiner's eye. The "localiser" is free from this defect, and also has the advantages that it can measure the interpupillary distance in squinters just as easily as in ordinary cases, and can measure the distance of each eye from the middle of the nose, and the elevation of one pupil over the other. It is made in metal to fit the trial frame, and is placed just before one eye and then another, enabling the observer to "sight" the patient's pupil as along a gun, while his own pupil is looked at by the patient. A slight modification of this instrument is now made, since it has occurred to the writer to utilise an ophthalmoscopic reflection from the cornea for measuring the distance between the visual lines, this reflection appearing to him to afford by far the most rational landmark for the purpose, since it marks out more closely than any other the very spot of the cornea traversed by the visual axis. All errors from high angle gamma, too, are thus avoided.[1] All that is necessary is to adjust the trial frame with the localiser in it, till the reflection, the notch, and the point are all in a line. It is only for strong lenses and special cases that such accuracy is required, and for ordinary work the usual pupillometers suffice.

Refractive Headaches.—The effort of accommodation in H, or its embarrassment in As, is very apt to give rise to a species of "referred pain" of a dull and continuous character, which may vary from a slight heaviness over the eyebrows to a more distressing widespread headache. In either case an area of tenderness may be found, sometimes, if sought for, after reading, just over the root of the nose. Sometimes the headache itself is confined to the same spot, but more often it is over each eyebrow. Occipital headache is by no means a rare accompaniment, but more generally implies either fatigue of the cerebral visual centres or defects of the extra-ocular muscles. Headache in one or both temples, or at the vertex, is more rarely of ophthalmic origin.

Refractive headaches are at first only disposed to occur towards the end of long spells of reading or fine work, but when more pronounced the subject wakes with them in the early morning. Gradual relief sets in during the morning hours, but evening work again aggravates the discomfort. In contrast to this daily headache we often meet with cases in which migrainous headaches, more or less periodical, are set up by refractive errors, with freedom from any pronounced headache in the intervals. Headaches sometimes cease entirely from the first day the correcting glasses are adopted, but more frequently it takes a few weeks to get sufficiently accustomed to their novelty to obtain their full benefit.

Simple myopia never causes headaches, though a myopic eye is sometimes the seat of congestive neuralgia. A curious feature about astigmatism is that low degrees are just as liable to cause headaches as the higher degrees, and especially if the principal meridians are oblique, or if the horizontal meridian of the cornea is more strongly curved than the vertical. It often only begins to give trouble about the age of thirty, and refractive headaches nearly always begin to die away after the age of fifty. "Headache spectacles" should be worn continuously to get the best effect.

Refrigerants.—Cooling drinks, such as lemonade, imperial, etc., used in fevers and febrile conditions to relieve thirst. *See* Pharmacology.

Refrigeration.—A means of preserving food-stuffs, such as meat, by freezing.

Refuse. *See* Sewage and Drainage (*Excrement and Refuse Disposal*).

Regeneration.—The restoration or repair of lost parts such as nerve. *See* Physiology, Tissues (*Neurons, Nutrition of*).

Regimen.—A system of rules as to diet and hygiene imposed upon a sick person (Lat. *regimen*, government).

Regio or Region.—A part or district of the abdomen, such as one of the abdominal areas (epigastric, umbilical, hypogastric, etc.). *See* Gynæcology, Diagnosis in (*Inspection of Abdomen, Regions of*).

[1] The angle gamma is the angle between the fixation line and the geometrical axis of the eyeball. It is greatest in hypermetropia, and least in myopia.

Regional Anatomy.—The study of the anatomy of the various regions of the body as regards the relations of the structures in any given region to each other.

Registration. *See* VITAL STATISTICS (*Births, Deaths, Sickness*).

Regius. — Literally the term means "royal"; *e.g.* a *regius chair, i.e.* a professorship having a royal endowment.

Regnault's Hygrometer.—An apparatus for ascertaining the dew-point. *See* METEOROLOGY (*Hygrometry, Dew*).

Regulation of Heat. *See* PHYSIOLOGY, NUTRITION (*Absorption of Food, Production and Regulation of Temperature*).

Regurgitation. *See* HEART, MYOCARDIUM AND ENDOCARDIUM (*Physical Signs of Different Forms of Heart Disease, Aortic and Mitral Incompetence*).

Reichert's Cartilage.—The hyoid bar of the second visceral arch of the embryo.

Reichert's Method.—A means of demonstrating the crystals of oxyhæmoglobin in the blood by laking it with ether and adding from 1 to 5 per cent of oxalate of ammonium.

Reichl's Test.—A method of detecting the presence of proteids in solution by adding alcoholic solution of benzaldehyde, sulphuric acid, and solution of ferric sulphate, producing a deep blue colour.

Reichmann's Disease.—A form of dyspepsia in which there is an intermittent or continuous increased secretion of gastric juice, generally hyperacid in reaction, irrespective of the presence of food in the stomach. *See* STOMACH AND DUODENUM (*Special Symptomatology of Gastric Neuroses*).

Reid's Line.—An imaginary line drawn backwards from the lower margin of the orbit through the centre of the external auditory meatus is called Reid's base line; it is used in cranio-cerebral topography, as are also two vertical lines related to it.

Reil, Island of.—One of the lobes of the cerebrum, lying on a deeper plane than the others, and hidden by the opercula, but visible if the fissure of Sylvius be opened up. *See* BRAIN, PHYSIOLOGY OF (*Anatomical*).

Reinsch's Test. — *See* TOXICOLOGY (*Arsenic, Tests*).

Reissner's Membrane. — A thin membrane in the membranous labyrinth of the ear, limiting the ductus cochlearis; membrana vestibularis.

Relapsing Fever.

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See also PAROTID GLAND, DISORDERS OF (*Parotitis, a Complication of Relapsing Fever*); MICRO-ORGANISMS (*Bacteria, Spirillum*).

SYNONYMS.—*Typhus recurrens, Famine fever, Spirillum fever;* French, *Fièvre à rechute;* German, *Armen-typhus.*

DEFINITION.—An acute infectious fever, characterised by the presence of a spiral micro-organism in the blood, by a rapid onset and defervescence, and by a repetition of the fever after a week's apyrexia.

HISTORY.—There seems to be no doubt that this fever is one of the diseases described by Hippocrates, although his description would appear to include cases which may, with more probability, be referred to other conditions. Singularly enough, however, it is not till 1739 that we find an account of an epidemic of relapsing fever, Rutty at that date giving an accurate description of it, though he did not distinguish it definitely from other fevers prevalent in Dublin at that time. The disease seems to have been frequently prevalent in Ireland, and particularly so in the first sixteen years of the nineteenth century. In most cases its epidemic prevalence coincided with that of typhus, a fact which prevented a definite distinction between the two fevers being made till Henderson, of Edinburgh, finally separated them in 1843, after both diseases had been epidemic for over two years. Since that time, as far as this country is concerned, relapsing fever has been of rare occurrence, the last cases in Great Britain occurring in 1869-70, although cases are reported as having occurred in Ireland since that date.

As regards *geographical range*, the fever may apparently exist in any climate. It has been during the nineteenth century fairly common in Northern Europe, very prevalent in Silesia, and is still to be found in Russia. It has also been of frequent occurrence in the famine districts of India. On various occasions it has appeared in North America, and its presence has also been noted in Algeria.

ETIOLOGY.—Relapsing fever has a particular interest in that it is caused by a micro-organism belonging to the group of protozoa. This organism is a spirillum, long and slender, and of varying length (16 to 40 μ), pointed at the ends, and, as Koch has succeeded in demonstrating, provided with true flagella. In fresh blood

it is actively motile, and attention may be drawn to it, on microscopical examination, by the manner in which the blood corpuscles are moved violently aside by its rapid movements. It stains readily with the ordinary basic aniline dyes, and may be searched for either in fresh blood, or by fixing blood on a cover-glass in the ordinary manner, washing in 1 per cent acetic acid, and afterwards in water, and then staining with gentian violet.

The spirillum is present in the blood during the whole of the primary attack, and also during the relapses. In the apyrexial intervals, however, it is not to be found, but the experiments of Metschnikoff have proved that they may, during this period, be present in masses in the spleen.

The vitality of the spirilla may be preserved for a considerable period, provided they are not exposed to heat, as a comparatively low temperature is sufficient to destroy them. All attempts to cultivate them on artificial media have failed, and the inoculation experiments performed on monkeys have been done with the blood of a patient suffering from the disease. Monkeys so inoculated develop large numbers of spirilla in the blood, and have a fever of short duration, terminating by crisis, but not followed by a relapse. The evidence at present is distinctly in favour of the spirillum which is known as the *Spirochæte Obermeieri* (its discoverer in 1873) being the real cause of the disease.

The more *remote causes* which favour the occurrence of an outbreak of relapsing fever, and increase the chances of its spread, are said to be destitution and famine. Indeed the latter condition has given the fever one of its most frequently used names. Overcrowding naturally favours the spread of any infective disease, and as this fever has been most frequently observed among the poorer classes, it is only natural to find conditions of overcrowding almost always associated with epidemics. Insufficiency of food may only be a cause in that it naturally weakens the resistance of the exposed person. In the outbreak in 1869 both Rabagliati in Bradford and Muirhead in Edinburgh noted that their patients were all sufficiently well nourished. All agree that, for some reason or other, males are more frequently affected than females. As regards age, the fever is a disease of early life.

Mode of Infection. — The most common method by which infection occurs is direct contact with a person suffering from the disease. Entering a badly ventilated room in which a patient is lying is sufficient to cause infection. As in typhus, therefore, infection would appear to be, to some extent, conveyed by the air, and to be contained in the exhalations of the patient. Murchison has noted that women working in hospital laundries frequently contract the disease, and we may therefore assume that the poison may be carried by fomites.

Morbid Anatomy.—Except for the usual microscopic conditions resulting from pyrexia, there is nothing to be found on post-mortem examination. Nothing characteristic or pathognomonic has been observed in fatal cases. The spleen is always much enlarged.

Symptoms and Course.—*The period of incubation* appears to be very variable. In certain cases the disease seems to develop within a few hours of exposure On an average five to ten days may be taken as the most probable time, though occasionally the period is prolonged to a fortnight.

The first paroxysm commences abruptly, the patient being suddenly seized with rigors, often severe, pains in the back and limbs, headache most marked in the vertex and occiput, and by a marked sensation of giddiness. This last symptom usually compels him to take to his bed at once. The temperature rises rapidly, and in a few hours may rise to anything between 102 and 106 degrees, even higher figures being occasionally recorded. The skin is dry and hot, and the patient at this stage is usually flushed, though even in the first twenty-four hours the skin may present the yellow tinge which is characteristic of this fever. The pulse is rapid, and is usually raised above 110, occasionally reaching 140 beats per minute before the end of the second day. As the fever progresses the tongue becomes covered with a thick yellow fur, but in the majority of cases it remains moist. Delirium may set in early, but is as a rule delayed for two or three days.

The temperature remains high during the paroxysm, morning remissions being only slight. A level of about 104° seems a fair average. The respiration becomes hurried, and frequently exceeds thirty. Cough is a common symptom, and auscultation reveals rhonchi and harsh breathing. The patient is tormented by excessive thirst, and frequently suffers from severe and persisting vomiting of greenish yellow matter. Jaundice is common, and nearly every case presents a distinct icteric tinge in the conjunctiva. The liver is enlarged and tender, and the spleen is also increased in size. The bowels are as a rule constipated. In bad cases the patient may be congested, or livid in the face. A certain percentage of cases, apparently, show a rose-rash on the trunk and limbs. The spots resemble small typhoid spots, though their distribution is more apt to suggest typhus. They do not disappear on pressure.

After from five to seven days of continued fever the paroxysm terminates as abruptly as it started, by a remarkably sharp *crisis*. It may be ushered in by slight diarrhœa, and is always accompanied by very profuse sweating. The fall of the temperature may be as much as 10° Fahr. in twenty-four hours, and may

continue till the thermometer registers only 94 or even 92 degrees.

After the crisis the patient enters on the "*first period of apyrexia*," or the "intermission." All the symptoms of the previous week have disappeared. A man who a day or two previously has been absolutely helpless, delirious, possibly in the "typhoid state," declares himself quite well, and is with difficulty persuaded to remain in bed. But on about the fourteenth day of the disease, that is to say, roughly speaking, a week after the crisis, the relapse starts as suddenly as did the original fever.

The *relapse*, or second paroxysm, except that it is usually a day or two shorter in duration, is simply a repetition of the first paroxysm. Once again the fever commences with rigors, headache, and giddiness, and the temperature rapidly rises. The pulse is often, if anything, more rapid than in the first attack, but this appears to have little significance, Rabagliati having noted pulses of even 160 in favourable cases. The thirst, jaundice, and vomiting may be as bad as ever, but, broadly speaking, the relapse is, as a rule, slightly less severe than the first paroxysm. The patient usually voids a greater quantity of urine than in his first attack, and this increase is most frequently noticed just before the crisis, one of Muirhead's patients having passed 84 oz. in the twenty-four hours.

The second crisis is in every way comparable to the first. The temperature falls with equal rapidity, and the sweating may be even more severe, sudaminal rashes being of common occurrence. Subnormal temperature for some days after is the rule, and there may be a considerable amount of prostration.

The *second period of apyrexia*, which usually coincides with the *period of convalescence*, is now reached. Occasionally there may be, however, a second or even a third relapse. If such is the case, the symptoms are, as a rule, milder. The convalescence is at times protracted and interrupted by complications.

Complications and Sequelæ.—Pharyngitis is a common complication. Vomiting and diarrhœa may be so severe as to assume the importance of complications. Bronchitis is not uncommon, and occasionally pneumonia has been noticed. Œdema glottidis and laryngitis are also met with. Epistaxis is frequently observed. Delirium is most frequently seen just before the crisis, but occasionally persists through the whole fever. Insomnia is of common occurrence. The most important sequela is ophthalmia, which seems very liable to occur in convalescence. Glandular enlargements in the neck, groin, and parotid are not unusual.

Diagnosis.—The most certain method is microscopic, the detection of the spirillum settling the question finally. Apart from this, diagnosis depends on the presence of an epidemic, on a sudden onset of high fever, with giddiness, slight jaundice, and vomiting, and on the marked crisis. The fever is most likely to be confused with influenza, typhus, and malaria. The occurrence of the relapse removes any difficulty.

Prognosis.—This may be said to be uniformly favourable except in old persons and complicated cases. The death-rate seldom exceeds 6 per cent. Severe diarrhœa, marked jaundice, and excessive prostration are the worst symptoms.

Treatment.—It seems to be generally agreed that there is nothing which influences the course of relapsing fever. All that can be done is to treat symptoms and complications as they arise. A tonic treatment with strychnine or nux vomica has been recommended. Insomnia should be appropriately combated. Quinine and other drugs have been tried without success with a view of preventing the relapse.

Relaxation.—Reduction of tension or contraction (*e.g.* muscular), or loosening (*e.g.* relaxation of the pelvic joints).

Religious Insanity. *See* Insanity, Nature and Symptoms (*Systematic Delusions, Religious*).

Remak's Ganglion.—A plexus of nerves in the wall of the sinus venosus of the heart. *See* Physiology, Circulation (*Heart, Intra-cardiac Mechanism*).

Remak's Sign.—The double sensation resulting from the prick of a pin, experienced in tabes dorsalis.

Remak's Type (of Paralysis).—Paralysis affecting the muscles of the arm (deltoid, biceps, brachialis anticus, supinator longus).

Remission.—The period during which there is a temporary abatement of the symptoms of a disease, especially of the fever accompanying malaria.

Remittent Fever. *See* Malaria (*Nomenclature*); Tropics, Unclassed Fevers of (*Non-Malarial Remittent Fever*); Undulant Fever (*Synonyms*).

Remote Action.—The indirect or secondary effect of a drug, that which is the result of the primary or direct action of it; thus convulsions are a remote effect of curare, the drug causing in the first place paralysis of the respiration.

Renal.—Relating to the kidneys; *e.g. renal asthma, renal calculi, renal cirrhosis*, etc. *See* Kidney, Physiology of; Kidney, Surgical

Affections of; Nephritis. *See also* Alcohol (*Special Physiology, Renal Functions*); Alcohol (*Indications, Renal Diseases*); Anæsthesia, Ether (*Ether and Renal Disease*); Angina Pectoris (*Diagnosis, Renal Calculus*); Appendix Vermiformis (*Appendicitis, Diagnosis*); Ascites (*Causation*); Asthma (*Origin*); Children, Clinical Examination of (*Urinary System*); Colic (*Diagnosis*); Gall-Bladder and Bile Ducts, Diseases of (*New Growths, Diagnosis*); Gangrene (*Septic*); Gout (*Irregular, Renal System*); Hæmaturia; Hydropathy (*Chronic Diseases, Bright*); Hysteria (*Disorders of Urinary System*); Ovaries, Diseases of (*Tumours, Diagnosis*); Retina and Optic Nerve (*State of Retinal Vessels in Renal Disease*); Spleen, Surgery of (*Wandering Spleen, Diagnosis*); Stomach and Duodenum, Diseases of (*General Etiology, Renal Causes*); Stools, Intestinal Sand (*Diagnosis*); Tabes Dorsalis (*Symptomatology, Renal Crises*); Therapeutics, Health Resorts (*Climate, Effect on Kidney Disease*); Typhoid Fever (*Complications, Urino-Genital*); Unconsciousness; Urine, Pathological Changes in; Visceral Pain (*Radiation*).

Rendu's Type.—A type of tremor met with in hysteria; it is increased by volitional movements.

Reniform.—Kidney-shaped.

Renipuncture.—Puncture of the capsule of the kidney. *See* Decapsulation.

Renk's Method.—A method of estimating the amount of ground air in the soil, by placing the soil (measured quantity) in a graduated glass vessel, covering it with water, and shaking it till the air is all expelled, when, from the level at which the water stands, the amount of air expelled can be estimated.

Rennes-les-Bains. *See* Balneology (*France, Chalybeate*).

Rennet. *See* Physiology, Food and Digestion (*Food Supply, Milk*).

Rennin. *See* Physiology, Food and Digestion (*Stomach, Secretion, Milk-curdling Enzyme*); Digestion and Metabolism (*Gastric Juice*); Enzymes.

Repair. *See* Wounds (*Mode of Repair*).

Repercolation.—A stage in the process of percolation (*q.v.*).

Repercussion.—Ballottement in the diagnosis of pregnancy; literally, striking back again. *See* Pregnancy, Diagnosis.

Reposition.—Replacement, *e.g.* of the retroverted, inverted, or prolapsed uterus or cord. *See* Labour, Accidental Complications (*Prolapse of Cord, Treatment*); Labour, Injuries to Generative Organs (*Inversion of Uterus, Treatment*); Pregnancy, Affections of Generative Organs (*Displacements of Gravid Uterus, Prolapsus, in Hernial Sac, Retroversion*, etc.); Uterus, Displacements of the (*Treatment*).

Repositor.—An instrument for replacing an inverted uterus or a prolapsed cord, etc.

Reproductive Organs. *See* Generation, Female Organs of; Ovaries; Penis; Scrotum and Testicles; Uterus; etc., with their *Cross References*.

Reptilia. *See* Heart, Physiology of (*Comparative Anatomy, Reptilian*); Heart, Congenital Malformations of (*Defect of Interventricular Septum*).

Resection.—An operation in which parts are cut out; *e.g.* resection of a joint, submucous resection of the nasal septum, etc. *See* Joints, Diseases of (*Tuberculous Disease, Operative Treatment*).

Reserve Air. *See* Physiology, Respiration (*Amount of Air Respired*); Respiration (*Physiology*).

Residual Air. *See* Physiology, Respiration (*Amount of Air Respired*); Respiration (*Physiology*).

Resin.—Resina or rosin is the residue left after crude turpentine has had the oil of turpentine distilled from it; it contains abietic acid (*q.v.*), and there are two official preparations (*Emplastrum Resinæ* and *Unguentum Resinæ*); it is used as an application for indolent ulcers and wounds, sometimes in the form of a soap. *See also* Oils; Oleo-Resins.

Resistance. *See* Electricity; Immunity (*Power of Resistance to Bacteria*).

Resonance. *See* Chest, Clinical Investigation of (*Percussion, Auscultation*); Lung, Tuberculosis of (*Physical Signs*).

Resorcin.—A colourless crystalline antiseptic ($C_6H_4(OH)_2$), so called because it was originally obtained from resin and because it resembled orcin; it is a dihydroxybenzene, and is isomeric with pyrocatechin and hydroquinone; it is chiefly used as an ointment in chronic skin diseases (*e.g.* eczema).

Respiration.

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See also Alcohol (*Special Physiology, Respiration*); Anæsthesia (*Arrest of Respiration*); Asphyxia (*Resuscitation*); Asthma (*Symptoms*); Brain, Affections of Blood-Vessels (*Anæmia, Symptoms; Occlusion of Vessels, Symptoms; Cheyne-Stokes Respiration*); Brain, Surgery of (*Compression, Symptoms; Cerebral Abscess, Symptoms*); Brain, Tumours (*Localising Symptoms, Respiration*); Chest, Clinical Investigation of (*Respiration*); Children, Clinical Examination of (*Respiratory System*); Climate (*Effect on Respiration*); Hæmoptysis (*Treatment, Artificial Respiration*); Hemiplegia (*Respiratory Movements*); Hysteria (*Disorders of Respiratory Organs*); Labour, Stages and Duration (*Second Stage, Phenomena*); Larynx (*Laryngeal Stridor and Laryngismus Stridulus*); Lungs, Diseases of; Lymphatic System (*Status Lymphaticus*); Meningitis, Tuberculous (*Respiratory Symptoms*); Meteorology (*Influence on Health*); Physiology, Respiration; Pneumonia (*Symptoms*); Pulse; Shock (*Treatment*); Stammering (*Treatment*); etc.

Absorption of oxygen and elimination of carbon dioxide, two conditions essential for the maintenance of the vitality of every animal organism, together constitute the process of respiration. In all the higher animals there is (*a*) external respiration, the interchange between the gases of the blood and those of the water or air, whereby O is absorbed and CO_2 eliminated, and (*b*) internal respiration, the interchange between the gases of the blood and lymph on the one hand and those of the tissues on the other, the blood thereby gaining CO_2 and parting with O. In man the lungs are the chief seat at which O is taken into the organism and at which CO_2 is eliminated.

The visceral layer of the pleura, covering the outer surface of the lung, is in intimate contact with the parietal pleura forming the innermost layer of the thoracic wall. The lungs themselves are highly elastic, and from the time of birth onwards are distended with air. But if air enter the pleural cavity—in which there is normally a state of negative pressure—the lung collapses or recoils in consequence of its elasticity, and part of the thorax, formerly filled by distended lung, is then filled with air.

In respiration there is an alternating indrawing of air into and expulsion of air from the lungs, these processes being termed respectively inspiration and expiration.

Inspiration.—As the intrapulmonary pressure is reduced by any enlargement of the thoracic capacity, each thoracic expansion will be followed by the drawing of air through the trachea and bronchi into the lungs. Inspiration is the result of muscular action; contraction and consequent descent of the diaphragm increases the vertical diameter of the thorax, the transverse diameter of which is enlarged by the elevation and rotation outwards of the ribs, whilst an increase of the antero-posterior diameter is due to a movement upwards and outwards of the ribs, costal cartilages, and, it may be, of the sternum also. The chief muscles of ordinary inspiration are, apart from the diaphragm, the external intercostals, which, on contraction, elevate all the ribs except the first. Less important inspiratory muscles are the intercartilaginous portions of the internal intercostals, the scalenus anticus, serratus posticus superior, and the levatores costarum. Many other muscles may act as muscles of forced inspiration, but of these the most important are the sterno-mastoids, scaleni, trapezei, and pectoral muscles. Thoracic expansion during inspiration is not of uniform character in all persons. In young children inspiration is mainly caused by the diaphragm, the type of respiration being termed abdominal; in men respiration is mainly of the diaphragmatic or abdominal type, whereas in women the inspiratory expansion is most marked in the upper part of the thorax, this being known as thoracic type of respiration.

Expiration under ordinary circumstances is a purely passive movement. The action of the inspiratory muscles having ceased, the distended lung partially collapses, and in doing so draws inwards the thoracic wall, which moreover tends to return to the state prior to the inspiratory movement owing to the elastic recoil of the costal cartilages, to the action of gravity acting on the chest wall, and also to retraction of the abdominal muscles. In emphysema, deficiency or loss of the pulmonary elastic tissue is one of the main causes of the difficulty in expiration, and of the consequent permanent over-distension of the lungs. Forced expiration is effected by various muscles, of which the most important are the muscles of the abdominal wall; yet all muscles which depress the ribs or compress the abdominal viscera, the serratus posticus inferior, the interosseous portions of the internal intercostals and the sacro-lumbalis, for example, are accessory muscles of expiration.

Respiratory Rhythm and Rate.—In normal respiration, inspiration and expiration succeed each other rhythmically, and the length of time occupied by inspiration in relation to that of expiration is on an average represented by the ratio 5 : 6; whilst after the completion of expiration there is a short pause. The most marked deviation from the normal respiratory rhythm is seen in Cheyne-Stokes respiration (*see* p. 509). In man the number of respiratory movements per minute is about 14-20; but even within physiological limits the frequency of respiration varies greatly in accordance with such factors as age, posture, exercise, and so forth, the frequency being, for example, greater in infancy and childhood than during adult life, and being decreased in any individual when he assumes the recumbent posture.

The volume of air inspired during each ordinary inspiration, *tidal* air as it is termed, amounts to about 500 c.cm., or 30 cubic inches. *Complemental* air is the further volume which by the deepest inspiration can be indrawn after an ordinary inspiration; *supplemental* or *reserve* air is the volume which can be forcibly expelled after ordinary expiration is completed; *residual* air is that remaining within the lungs after expulsion of the reserve air. The *vital* or *respiratory capacity* of an individual is the volume of air which he can expel after as forcible an inspiration as possible. The vital capacity varies with the sex, age, height, and weight of the individual, but averages about 3500 c.cm. to 4000 c.cm.

Changes in Respired Air.—The atmosphere consists of 20·96 volumes per cent of oxygen, about 79·0 volumes per cent of nitrogen, and 0·03 volumes per cent of carbon dioxide; further, the inspired air contains a variable quantity of watery vapour, usually contains dust particles, and always contains micro-organisms in greater or lesser numbers (*see* p. 510). The air, on passing through the nose, trachea, and bronchi, gains moisture, and its temperature is raised, and by diffusion its gases then mix with those of the air persisting in the pulmonary alveoli. The expired air, as compared with that inspired, is warmer, poorer in oxygen by about 4 per cent, but richer by about 4 per cent in carbon dioxide, and richer in aqueous vapour. The chief of these alterations are consequent on an interchange between the gases of the alveolar air and those of the blood in the pulmonary capillaries. The arterial blood of man contains about 21 volumes per cent of oxygen, and 40 volumes per cent of carbon dioxide, whilst venous blood coming to the lungs contains about 7-12 volumes per cent less O, and about 8 volumes per cent more CO_2. It is therefore evident that the blood in passing through the lungs gains oxygen and parts with CO_2, and this interchange of gases is probably largely due to gaseous diffusion. For the tension of oxygen in alveolar air is greater than that of the oxygen in venous blood, whereas the tension of carbon dioxide in venous blood is greater than that of the carbon dioxide in alveolar air. The oxygen is, however, not merely dissolved in the blood, but is in chemical combination with hæmoglobin, and, moreover, there are probably other factors besides diffusion concerned in the gaseous interchange.

The Nervous Mechanism of Respiration.—The special respiratory muscles are innervated from different parts of the central nervous system. The diaphragm is innervated through the phrenic nerves, the intercostal muscles through the intercostal nerves, the muscles which open the glottis through the inferior laryngeal branches of the vagus, and the muscles of the alæ nasi through the facial nerves. The impulses transmitted through these nerves are dependent upon and co-ordinated by the respiratory centre which is situated in the medulla oblongata. The precise situation in the medulla of the cells constituting this centre is a matter of some dubiety; Flourens in 1824 regarded the centre as being located just below the apex of the calamus scriptorius, but the observations of later investigators indicate that the cells forming the centre lie in the formatio reticularis on both sides of the median line.

The activity of the centre is automatic; it can act independently of any afferent impulses. It is generally supposed that the automatic activity of the centre is dependent on stimuli derived from the blood, in that the cells are stimulated to activity by a venous condition of the blood, but whether excess of carbon dioxide or deficiency of oxygen be the determining factor is as yet undecided. The centre may, however, be stimulated reflexly by afferent impulses, and that the centre is constantly being influenced by such stimuli is shown by the fact that although when the centre is cut off from all afferent impulses, respiration continues, it consists in rhythmic spasmodic movements the frequency of which gradually diminishes till the movements finally cease entirely. Of all afferent impulses acting on the respiratory centre the most important are those transmitted by the vagi, these nerves being essential for the normal respiratory rhythm, and being the sole nerves whereby afferent stimuli are constantly reaching the respiratory centre. After division of both vagi, the respiratory movements become markedly slow and deep, and it has been determined that the nature of the vagus influence is that of accelerating and shortening the amplitude of the respiratory movements, this being effected by the inhibitory impulses produced by distension and collapse of the lung.

When the *superior laryngeal nerve* is stimulated there is inhibition of the inspiratory movement, and with a stronger stimulus forced expiratory movements are induced.

Stimulation of the *glosso-pharyngeal nerve* results in transient cessation of respiration. This is a constant phenomenon during the act of deglutition.

Stimulation of any ordinary sensory nerve is usually followed by acceleration and lengthening of inspiration, a well-known fact of considerable clinical importance.

The influence exerted on the respiratory centre by the higher parts of the brain is as yet imperfectly determined. Still it is improbable that in a normal state such impulses are constantly in action, for if, the vagi being intact, a section be made through the upper level of the medulla, respiration continues in a normal manner. If, however, the vagi also be divided, the normal respiratory rhythm is markedly affected, long inspiratory movements alternating

with prolonged pauses, and the animal finally dies of asphyxia.

If from any cause there be interference with the respiratory movements, or if there be obstruction to the pulmonary circulation, or should there be any other cause, such as anæmia, for example, interfering with the interchange between the gases of the alveolar air and those of the blood, the resulting venous state of the blood increases the activity of the respiratory centre. The rate and depth of the respiratory movements are then increased, and the condition is known as that of *hyperpnœa*. Should the venous state of the blood increase in intensity, the respiration becomes more markedly difficult, and there is a condition of *dyspnœa* (*q.v.*), which, should the cause be sufficiently great and its period of duration sufficiently prolonged, passes into the state of *asphyxia* (*vide* vol. i. p. 301, and vol. vi. p. 259), and this in turn may cause death.

In contrast to dyspnœa there is the condition termed *apnœa*, in which the respiratory movements are rendered shallower than normal, or brought to an entire standstill as the result of repeated, forcible inspirations. Unless the vagi be divided, apnœa is not due to excessive oxygenation of the blood; when these nerves are intact the apnœa is caused by the repeated inspirations stimulating the inhibitory fibres of the vagi.

The *muscle fibres of the bronchi* are innervated by motor fibres of the vagus. Stimulation of the peripheral end of either vagus nerve results in constriction of the bronchioles of both lungs, whilst if the vagi be intact, stimulation of sensory nerves also causes reflex contraction of the bronchial muscles. Spasm of the bronchial muscles probably causes less difficulty in the entrance of air through the bronchioles than in exit of air through them, and such spasm is the most uniformly accepted explanation regarding the nature of asthma (*q.v.*).

Cheyne-Stokes Respiration. — In disease considerable variations from the normal respiratory rhythm are not uncommon. Firstly, there may be undue prolongation of inspiration or of expiration, and a condition of inspiratory or of expiratory dyspnœa (*q.v.*). Again, the respirations may be irregular, and the irregularity may be rhythmical or arhythmical in character. Respiration of the latter type is common in association with cerebral affections. In cases of cerebral hæmorrhage or tumour, for instance, the character of the respiration may be irregular, as well as slow and sighing or stertorous; whilst in the later stages of meningitis there is often an arhythmical irregularity, combined with rapidity and shallowness of the breathing.

Of rhythmical irregularity there are various varieties. Thus the respiration may be intermittent, the natural rhythm being at regular intervals suddenly interrupted by a complete pause. But the most striking variety of rhythmical irregularity is that termed *Cheyne-Stokes respiration*. In this type there are two alternating phases, the first one being that of apnœa, and there being a complete cessation of respiratory movements, and the second being a period of gradually increasing and then gradually decreasing respiratory activity. At the commencement of this latter phase the respiratory movements are feeble, faint, and shallow, but each successive respiration becomes fuller and more rapid till a maximum of forcible inspiration and expiration is attained, whereupon the rate and force of the subsequent respiratory movements gradually decrease until the breathing becomes so feeble as to be barely appreciable, and, finally, there is complete cessation of respiration. The cessation of respiratory movement, or the period of apnœa, having lasted for a variable time, the respirations commence once more in the manner described. The duration of the respiratory pause is usually about twenty, rarely more than thirty seconds, the period of activity lasts as a rule about forty or forty-five seconds, and the whole cycle extends over a period of a minute or rather more. The number of respirations occurring during the period of activity is subject to variation, though usually about fifteen or twenty. The above description is that of typical Cheyne-Stokes breathing, but frequently rhythmical irregularities of respiration do not conform exactly to the type, and are to be regarded as varieties or modifications of Cheyne-Stokes respiration.

The onset of Cheyne-Stokes breathing may be sudden, as in cases of cerebral hæmorrhage or of head injury, but is usually gradual and insidious, being preceded by dyspnœa and a rhythmic irregularity of respiration. When once developed, however, it is most marked if the patient be kept thoroughly quiet and at rest, or if he be asleep, and still more so if he be unconscious.

In some cases the pupil is contracted during the period of apnœa, dilated during that of dyspnœa, but in other instances the pupil is uniformly small. In some cases, further, there is an increase of blood pressure with retardation of the pulse apparent at the end of the respiratory pause, becoming more marked during the period in which the respirations are increasing in force, and disappearing as the respiratory movements become feebler.

Cheyne-Stokes respiration is seen as a symptom of various diseases. It is more frequently seen in association with *uræmia* than with any other condition; it may occur in cases of acute nephritis, but is exceedingly common as one of the uræmic phenomena in the late stages of chronic Bright's disease, whether the kidneys be the seat of chronic parenchymatous nephritis, or whether there be chronic interstitial changes with arterio-sclerosis.

It is also frequently observed in cerebral affections such as tumours, hæmorrhage, meningitis, etc.

Cheyne-Stokes respiration, again, is often developed in the course of *cardiac disease*, usually in cases of chronic valvular disease with associated myocardial degeneration and arteriosclerosis, or in cases of primary disease of the myocardium, and very rarely in cases of acute cardiac disease. In this group of cases the Cheyne-Stokes respiration is probably more directly due to the vascular disease than to any secondary implication of the kidneys.

Lastly, this form of respiration has been recorded in fatal cases of sunstroke, and in rare instances in connection with other conditions. Whatever be the morbid condition with which the Cheyne-Stokes respiration is associated, its occurrence is a matter of serious import; and though it does not invariably imply that disease is about to terminate fatally, yet the prognosis becomes more grave than it would otherwise have been.

The explanation of Cheyne-Stokes respiration has been the subject of much controversy. Traube's theory assumed that the excitability of the respiratory centre is reduced, and that CO_2 is present in excess in the blood. Filehne supposed that the CO_2 accumulating in the blood during the respiratory pause causes stimulation of the vaso-motor centre, and consequent arterial contraction. As a result the blood becomes still more venous, and the respiratory centre is then gradually awakened to activity, but oxygenation of the blood being effected the activity of the respiratory centre diminishes, and consequently the rate and force of the respiratory movements are reduced. According to Franck the irregularity of respiration is dependent on impairment or inhibition of the cerebral control exerted on the respiratory centre.

Bacteriological Aspects of Respiration.—As has been already stated, the air always contains micro-organisms. The majority of these are probably saprophytic and innocuous, and need not be considered further. Others, however, are pathogenic, and a few points regarding them are here dealt with. These bacteria exist in the air under two conditions: firstly, adherent to fine dust particles; and, secondly, within minute fluid droplets. In considering what risk an individual incurs by the respiration of air containing bacteria attached to particles of dust, it must be remembered that, although bacterial spores and tubercle bacilli withstand desiccation, most bacteria do not long maintain their vitality in the absence of moisture. The inhalation of a dust-laden atmosphere is in itself, therefore, probably capable of causing infection only when there are tubercle bacilli or spores, such as those of the *B. anthracis*, associated with the dust particles.

The minute fluid droplets which contain bacteria are probably more dangerous than solid particles. It was in 1897 that Flügge first demonstrated that phthisical individuals on coughing set free minute droplets of sputum which remain suspended in the air; and it was also proved that when any individual coughs, sneezes, or speaks, he causes the liberation from the secretions of the mouth, nose, or pharynx of minute fluid droplets which contain bacteria. These droplets are then in suspension in the air, and a current of air with a velocity of rather less than 0·1 mm. per second suffices to convey them still farther from the individual. Again, it is now known that such minute droplets, being inhaled, whether by the nose or mouth, may be carried down even into the finest bronchi by the current of inspired air. It is therefore evident that a patient affected with respiratory disease, such as tuberculosis, coryza, bronchitis, or influenza, disseminates in his vicinity droplets containing pathogenic bacteria; and it is also evident that not only are persons in the neighbourhood of that patient almost certainly inhaling those bacteria, but that the patient himself must inhale many of the bacteria which he has previously set free. It appears doubtful whether fluid droplets can be liberated from the tenacious sputum of ordinary croupous pneumonia.

But the mere inhalation of pathogenic bacteria does not suffice to cause infection, for the organism is well provided with means of protection. Large numbers of the inhaled bacteria will impinge against the mucous membrane of the nose and naso-pharynx, the former of which, at least, is known to possess marked bactericidal power; and such bacteria as are carried to the bronchi will likewise be destroyed, owing to the bactericidal power inherent in the bronchial mucous membrane, or having impinged on the cilia, will be eliminated during subsequent expiratory acts. The lower parts of the normal respiratory tract, therefore, are not invariably sterile, and may at times contain various micro-organisms. But as bacteria entering the healthy trachea or bronchi of man are probably quickly destroyed, these portions of the respiratory tract and also the pulmonary alveoli may, as a rule, be regarded as sterile, as in point of fact they are usually found to be in the smaller animals. If, however, there be a local predisposition to disease, or if the resistant power of the organism be reduced, pathogenic bacteria on being inspired will cause infection.

Rest-Cure.—The Weir Mitchell treatment. *See* Mitchell, Weir; Heart, Myocardium and Endocardium (*Treatment, Rest and Exercise*); Stomach and Duodenum, Diseases of (*Treatment of Gastric Neuroses*); Ulcers and Ulceration (*Treatment*).

Restiform.—Cord-like (Lat. *restis*, a rope), *e.g.* the *restiform body* of the medulla oblongata. *See* Physiology, Neuro-Muscular Mechanism (*Medulla Oblongata, Structure*).

Restitution, Movement of.—In obstetrics this term is applied to the movement which the head (or other presenting part) makes after its expulsion from the vulva; it carries the occiput back to the side of the maternal pelvis where it lay before internal rotation took place. *See* Labour, Diagnosis and Mechanism (*Presentations of the Vertex*).

Resuscitation. *See* Asphyxia (*Resuscitation*); Medicine, Forensic (*Deaths from Asphyxia*).

Retained Menses. — Cryptomenorrhœa. *See* Menstruation and its Disorders (*Retention of the Menses*).

Retching. *See* Anæsthesia, Chloroform (*Vomiting and Retching*); Emetics; Physiology, Food and Digestion (*Digestion in the Stomach, Vomiting*); etc.

Rete.—A net-like structure, such as the *rete testis* of the testicle and the *rete Malpighii* of the skin.

Retention.—The holding back of anything; *e.g.* the retention of urine (*see* Micturition), of the decidual membranes (*see* Abortion), of the dead fœtus (*see* Pregnancy, Intra-Uterine Diseases and Death), or of the placenta (*see* Labour, Retention of Placenta).

Reticulum.—A fine network; also, the second stomach of ruminants.

Retina and Optic Nerve.

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See also Amblyopia (*Toxic, Uræmia, Retinitis*); Brain, Physiology (*Optic Nerve*); Choroid, Diseases of (*Choroiditis, Diagnosis; Sarcoma, Diagnosis; Tubercular Disease; Retinal Separation*); Colour Vision (*Retinitis*); Diabetes Mellitus (*Symptoms of the Eye*); Eyeball, Injuries of (*Commotio Retinæ*); Glaucoma (*Secondary, Causes of*); Gout (*Irregular, Eye*); Malaria (*Pernicious Attacks, Retinal Hæmorrhages*); Nephritis (*Clinical Features, Chronic, Retinal Changes*); Ocular Muscles, Affections of (*Nystagmus, Causes*); Physiology, Neuro-Muscular Mechanism (*Vision, Stimulation of the Retina*); Pigments of the Body (*Melanins, Fuscin*); Pupil; Syphilis (*Tertiary, Eye Affections, In Children, Retinitis*); Trades, Dangerous (*Lead-Poisoning, Eye Changes*); Typhoid Fever (*Complications, Ocular*); Vision, Field of (*Retinitis Pigmentosa*); Vitreous Humour, Diseases of (*Hæmorrhage, Retinitis Proliferans*).

Affections of the Optic Nerve

Anatomy.—On leaving the eyeball the optic nerve courses backwards to the apex of the orbit, and then, passing through the optic canal, enters the cranial cavity. It now very soon joins its fellow of the opposite side, their junction forming the optic commissure or chiasma, where an important redistribution of nerve-fibres takes place. The newly distributed fibres, leaving the commissure, are again collected into bundles, now called the optic tracts, which pass backwards between the temporal lobes and the crura cerebri.

In the first and greater part of its orbital course the nerve is surrounded by fat, but deeper in the orbit it comes into closer proximity to the muscles arising from the apex, and at the optic foramen the nerve is in close relation with their tendons of origin.[1] About midway in its passage through the orbit the nerve receives the central vessels, which enter it from beneath and then run forward in its axis. Until it gains the interior of the cranial cavity the optic nerve is protected by a thick, loosely fitting outer sheath.

The ophthalmic artery accompanies the optic nerve in its passage through the optic canal, which latter affords little more than bare space for these structures to pass. It is important to observe that the bony plate forming the inner wall of the canal is very thin, and that this alone separates the optic nerve-sheath from the sphenoidal sinus.

The chiasma rests on the optic groove; above it the third ventricle is situated. The internal carotid arteries lie on either side, while close behind it is the pituitary body.

If we now examine the optic nerve more particularly, we find that in the optic canal and orbit it is surrounded by a tubular prolongation of the meninges, and we can recognise a dural, pial, and arachnoidal sheath. The dural sheath surrounds the nerve loosely, while the pial sheath is closely applied; the interval between these (intervaginal space) is subdivided by a fine prolongation of the arachnoid into two parts, subdural and subarachnoid, which communicate with the corresponding intracranial spaces. The highly vascular pial sheath sends processes inwards, which form a framework separating the bundles of nerve-fibres. Between each mesh of this framework and the nerve-

[1] These tendons of the superior and internal recti are closely incorporated with the optic nerve-sheath, and owing to this relation it has been asserted that movements of these muscles are particularly liable to cause pain in cases of retro-ocular neuritis implicating the nerve-sheaths in this locality.

fibres enclosed in it, there is a narrow interval occupied by lymph. By the lymphatics of the pial sheath, and by those which accompany the central vessels on their entrance into the nerve, there are communications between the lymph-spaces in the interior of the nerve and the intervaginal space. If we trace the nerve-sheaths forward to the eyeball, we find that the dural and arachnoidal sheaths here join and turn away from the nerve to be continued into the sclerotic, while the pial sheath is also united with the sclerotic and sends a few fibres into the choroid. The intravaginal space consequently ends abruptly in the sclerotic around the optic nerve entrance, and this has an important significance in pathological processes.

The intraocular end of the nerve is seen with the ophthalmoscope. It is situated to the nasal side of the centre of the fundus, and is known as the optic disc or papilla. The optic disc is nearly circular, but usually somewhat oval vertically; it is of a light orange-pink colour, with a characteristic superficial translucency. Its edge is traceable all round, but is most distinct outwards, and here the disc itself is also rather paler than elsewhere from the nerve-fibres and capillaries being fewer. About the centre of the disc we can observe a depression surrounded by the converging nerve-fibres; this is known as the physiological cup or pit, and frequently its floor presents a dappled appearance, grey spots separated by white interlacing lines. The white tissue or lamina cribrosa is the thick anterior end of the pial framework, while the grey marks are the meshes occupied by nerve-fibres. Also, near the centre of the disc the retinal blood-vessels first come into view, the arteries narrower and lighter in colour than the veins. On the disc the nerve-fibres are naked axis-cylinders, but they acquire a medullated sheath at the lamina cribrosa. In the rest of their course they are like the fibres of the white substance of brain and spinal cord; they are medullated and invested by neuroglia, but destitute of primitive sheath.

The functionally most important fibres are those coming from the ganglion cells around the fovea centralis (macular bundle). At the optic disc these fibres occupy the outer third, but after leaving the eyeball they soon gain a central position in the nerve-trunk, and continue as axial fibres of the nerve until it ends in the chiasma. Although arising from a small part of the retinal expansion, these macular fibres occupy a relatively large part of the nerve area (as seen on transverse section); this is due to the fact that each of these fibres represents the central connection of a single retinal element (cone), while each of the more peripheral optic nerve-fibres is connected with several retinal elements.

The other fibres, coming from the upper, lower, outer, and inner parts of the retina, occupy a corresponding area (upper, lower, etc.) of the optic nerve trunk. The most central fibres at the disc are those which come from the most peripheral parts of the retina, while the fibres situated next the pial sheath of the nerve-trunk come from the retina immediately around the disc. It is important to recollect this, for it shows that an enlargement of the normal "blind spot" will be the first result of an inflammatory or atrophic process affecting the fibres at the periphery of the nerve-trunk.

At the chiasma the optic nerve-fibres undergo a rearrangement, one important bundle being continued into the optic tract of the same side, while the remainder (constituting the larger bundle) cross over into the opposite tract. The non-crossing fibres correspond to those coming from the temporal half, and the crossing fibres to those coming from the nasal half of the same retina; the line dividing the retina into these two halves passes vertically through the macula lutea. Each tract, consequently, contains fibres from its own side (right or left) of both retinæ. It follows that a loss of function in one optic nerve will mean a visual failure confined to the same eye, while lesion of one tract is followed by visual failure of both eyes, but only of those fibres coming from the homonymous side of both (right or left), and will therefore be expressed by the loss of the opposite half-field (left or right homonymous hemianopia). Lesion of the chiasma may implicate fibres from one eye only, or from both; the most usual implication of the chiasma is central, affecting the crossing fibres from both nasal retinæ, so that bi-temporal hemianopia is the result (*see* article on "Vision, Field of").

Each optic nerve-fibre is the central process (axon) of a retinal ganglion cell, and is nutritionally dependent upon this cell. The conducting value of the fibre may consequently be impaired in two distinct ways—by an affection (*a*) of the ganglion retinæ, or (*b*) of the fibre itself in any part of its course.

Diseases.—Disease affecting the optic nerves or tracts produces symptoms that can be examined by different clinical methods, viz.:—

(1) *By testing vision.*—This test includes the examination of central and peripheral vision for form, colour, and light appreciation.

(2) *By examining the action of the pupil to light.*

(3) *By observation with the ophthalmoscope.*

In every case where optic nerve disease is looked for, all these methods must be employed. It will be sufficient here to mention that, in acute inflammatory conditions dependent upon intracranial disease, the ophthalmoscopic observation is the most important, while the visual test is particularly valuable in the early stages of implication of the nerve behind the eyeball of local origin.

General Pathology.—The optic nerve is

peculiarly prone to suffer in affections of the central nervous system, in some forms of poisoning, and in certain general disorders. Inflammation of an acute or chronic character may attack it at any point in its course, may affect its whole thickness or only a part of it, and may or may not lead to permanent atrophy, total or partial. Again, without recognised inflammation, the nerve may undergo degenerative changes resulting in a simple atrophy. Occasionally the occurrence of an inflammation or simple atrophy of the nerve is intelligible enough, as, *e.g.*, an optic neuritis in association with orbital cellulitis, or a simple atrophy subsequent to division of the nerve-trunk. Frequently, however, the pathological process is difficult to trace, and our knowledge still rests largely on hypothesis. It is impossible in the space at our disposal, and would be of doubtful benefit, to enter into discussions as to the causal relation between optic neuritis and intracranial disease, etc.; but the following facts should be remembered in connection with the exceptional vulnerability of this nerve:—

1. *From its development and structure the optic nerve is to be regarded as part of the central nervous system.*

This serves to explain its liability to degenerative changes, similar to those occurring in certain strands and fibres in the brain and spinal cord, in post-tertiary syphilis (tabetic atrophy), and to those occurring irregularly in disseminated sclerosis; and probably, also, this similarity of nerve-tissue is responsible for the occasional association of optic neuritis with acute myelitis, due presumably to the selective action of some toxine on these parts of the central nervous system.

2. *The sheaths of the nerve are direct prolongations of the cerebral meninges.*

We thus have the liability to a direct transmission of inflammation of the meninges along the nerve-sheaths to the fibrous framework of the nerve itself, which latter may be thus attacked in any part of its course, but particularly at the lamina cribrosa and at the entrance of the blood-vessels.

3. *The intervaginal spaces of the nerve are continuous with the meningeal cavities of the brain.*

In as far as these intervaginal spaces are also connected with each other, and with the lymphatic spaces surrounding the nerve-fibre bundles, it is obvious that fluid circulating in the lymphatic system of the nerve may contain inflammatory material primarily derived from within the cranium.

Again, serous fluid or blood effused into the meningeal cavities may find its way into the sheath-spaces and injure the nerve by direct pressure.

4. *The absence of primitive sheath probably renders the nerve-fibres more vulnerable in the retro-ocular portion; while, in the intraocular part of the nerve, the naked axis-cylinders are directly exposed to the action of the surrounding fluid.*

5. *It must be borne in mind that we can detect inflammatory and atrophic changes in this nerve such as might readily escape notice in other cranial nerves.*

Of less exceptional significance, but still important in considering the great vulnerability of the optic nerve, we would also mention:—

6. Their position, relation, and long course along the base of the brain render the nerves, commissure, and tracts liable to be affected in inflammation of the basal membranes, and to be damaged locally by pressure from new growths or from a distended third ventricle.

7. After entering the optic canal the nerve has for the rest of its course all the exposures of a peripheral nerve.

8. The narrowness of the optic canal must cause further constriction in an already existent œdema; and the disposition of the lamina cribrosa and of the nerve-sheaths where pierced by the central vessels will promote a similar effect by pressure on the blood-vessels and lymphatics.

Inflammation of the Optic Nerve.—The expression "optic neuritis" is the generic term for inflammation of any part of the optic nerve. When the intraocular end of the nerve or papilla is inflamed, the condition is called "papillitis"; while "retro-ocular neuritis" implies inflammation of the nerve behind the eyeball, without implication (at first at least) of the papilla. The terms "axial neuritis" and "perineuritis" are used to denote the parts of the nerve involved, such as would be seen as presented on cross section.

As a rule both nerves are affected, and similarly, though not necessarily to the same degree, nor exactly at the same time. *Unilateral* optic neuritis is generally dependent on a local cause, such, *e.g.*, as orbital cellulitis; but it may also occur in association with intracranial lesion.

Symptoms.—Optic neuritis may exist without there being any subjective symptoms directly indicating its presence—ocular pain, photophobia, and impairment of vision being alike absent. Photophobia, indeed, is rarely found as an accompaniment, and is never a prominent feature. Deep-seated pain in the orbit, and tenderness on pressure over the globe or on eye movement, are practically present only when the nerve is involved in an inflammatory condition affecting the orbital tissue surrounding the nerve. Vision may remain perfect for some time; indeed, in exceptional cases, papillitis from intracranial disease (gumma, for example) may occur, remain for some weeks, and disappear again, without sight ever being impaired.

Usually, however, failure of vision is a prominent symptom. This sometimes progresses rapidly, but blindness never occurs suddenly and remains permanently; although short, temporary attacks of darkness are not very uncommon in papillitis due to brain tumour.

It must be borne in mind that amblyopia occurring in a case of papillitis may be due, not to the neuritis, but to direct pressure of a tumour on nerve, chiasma, or some more central part of the visual tracts. The nature of the amblyopia (uniocular failure, bi-temporal loss of field, or homonymous hemianopia) will suggest this explanation, besides indicating the part involved, and may thus be of great localising value.

As a rule, in *papillitis* the failure of vision is gradual, slowly affecting central vision (especially at first for green and red), with progressive peripheral, sectorial, or irregular contraction of the visual field. Sometimes a neuritic sector-defect is binocular, simulating hemianopia of intracranial origin.

In *retro-ocular neuritis* the loss of vision is often rapid, and occurs mainly in the form of a central scotoma, at first only or mainly for green and red, later for all light (absolute scotoma). In acute cases of this kind there may be great amblyopia without any ophthalmoscopic change; indeed, in such cases the papilla is usually first involved when the severity of the attack is passing off and vision has begun to improve. This form is not infrequently one-sided, and the complaint of great loss of vision, while the optic disc appears normal, may excite the suspicion of malingering or of (so-called) hysterical amblyopia. In such a case of doubt an examination of the pupillary reaction will clear up the diagnosis; if the pupil of the (query) amblyopic eye fails to react as well as the other to direct stimulation by light, but acts perfectly consensually, we may rely upon there being some affection of the optic nerve to account for the blindness, although there is no other objective evidence of it.

Ophthalmoscopic Changes.—In well-marked papillitis the following appearances are found:—The edge of the optic disc is quite hidden from view, and the retina for some distance around is rather pale and cloudy. The general vascularity of the disc is much increased, while a greyish haze dulls its deeper details and obliterates the physiological cup. The whole papilla is swollen, so as to be considerably above the level of the adjoining retina, and many fine straight vessels may be seen radiating from it on every side, coursing over the obscured edge. The large retinal vessels, particularly the arteries, are often hidden at their exit on the papilla, and also here and there in their course over or just beyond it. The veins are enlarged, dark, and tortuous, showing evidently an obstructed venous flow. The arteries are normal or slightly narrowed. On the disc or in the adjoining retina hæmorrhages are often present, and sometimes also smooth, opaque, white spots.

In the slighter forms there may be merely some increased redness, doubtful swelling of the disc, with obscuration of the edge (particularly above and below), and distended veins.

Between these extremes there are many gradations observable in different cases, marked by more or less swelling and obscuration of details. These different degrees may simply be stages in the development of a gross papillitis, or any one may be the height of the affection where the papillitis is of less severity.

In a rapid, intense papillitis or "choked disc," the chief visible changes in the papilla (swelling and increased vascularity) are probably due mainly or entirely to œdema with pressure on the central vein, and in such cases there are generally evidences of great increase of intracranial pressure—intense headache with sickness. Should the intracranial pressure be relieved in such a case, the ophthalmoscopic signs quickly subside and leave no trace of their former presence.

As a rule, however, the progress of subsidence after papillitis is much more gradual, and of a less simple character. In such cases the redness of the papilla gradually disappears, while the swelling becomes reduced both in height and extent. The veins usually remain distended for some time, but ultimately all the vessels are narrowed. The edge of the disc again becomes visible, the outer margin coming first into view. The disc is opaque and woolly-looking at first, but gradually gets smoother; its surface is occupied by less or more newly formed tissue, which often permanently conceals the lamina cribrosa. Around the disc there is frequently a pale zone, sometimes with scattered pigment granules, the result of changes in the retinal pigment layer. Should complete atrophy result, the disc is usually of a brilliantly white colour, and the larger vessels on or near it are often bordered by opaque white lines.

In cases of retro-ocular neuritis, the resulting pallor of the disc is sometimes the first ophthalmoscopic sign of the disease, but generally it is preceded by papillitis, which may be slight, moderate, or intense. The amount of resulting atrophy is usually comparatively slight, but varies much in degree. Not infrequently the pallor of the disc is greatly in excess of what we should expect from the excellent recovery of vision.

Rapid failure of vision, usually in one eye only, often accompanied by pain and tenderness in the neighbourhood, absence of early ophthalmoscopic changes, and a tendency to recovery, are the usual prominent clinical features in retro-ocular neuritis.

Under this head it has also been usual to class "toxic amblyopia," but there are strong reasons for the belief that the affection of the optic nerve in these cases is in reality secondary to changes in the large retinal ganglion cells at the macula (*see* "RETINA," p. 520).

Pathological Anatomy.—In papillitis of intracranial origin the evidences of inflammation seldom extend beyond the lamina cribrosa. The early stage of a severe papillitis is unaccompanied by affection of the connective tissue, the changes merely consisting of venous hyperæmia, infiltration, œdematous and ganglionic thickening of the axis-cylinders. Later, there is a new formation of œdematous connective tissue in the substance of the papilla, and filling the physiological pit, with an exudation of white cells; while behind the lamina cribrosa the medullary sheath of the nerve-fibres is swollen and undergoing granular degeneration. When the stage of subsidence with atrophy is reached, we find much connective tissue, partly of new formation, partly due to hypertrophy of the pre-existing framework; the nerve-fibre bundles are thus reduced in size from pressure, and either degenerated or quite disintegrated and replaced by hyaline material; the walls of the arteries are thickened, with consequent contraction of their lumen, tending to partial or total obliteration of the smaller vessels.

In retro-ocular neuritis the pathological changes doubtless vary greatly, according to the nature of the attack, while opportunities for examining the nerve during the acute stage seldom occur. The connective tissue of the septa has been observed to be thickened and vascular, with consequent pressure on the nerve-fibres. In later stages, when the inflammation subsides, the nerve-fibre meshes may either be restored, with relief of pressure and return of function, or they may be further narrowed, with subsequent permanent atrophy of the contained nerve-fibres. The nerve is most liable to be attacked in the optic canal, or just behind the eyeball, and therefore the pathological changes are most marked in one or other of these localities. In either case it is the macular bundle that is mainly involved, thus accounting for the central scotoma clinically recognised.

Etiology.—The causes of optic neuritis may be divided broadly into two great classes, viz. those—

(1) *Of local influence, e.g.* orbital inflammation.

(2) *Of general influence, e.g.* renal disease and anæmia.

(1) *Local Causes.*—Under this heading are to be included cases of optic neuritis due to affections of the cranial bones, orbit, and cranial cavity.

(*a*) Of all the causes of optic neuritis, *intracranial disease* is much the most frequent.

Any *tumour* within the cranium may produce papillitis, irrespective of its nature, position, or size; but it is most apt to occur when the tumour is of rapid growth, and when it is situated at or near the base of the brain or in the cerebellum. It is almost a universal rule that both nerves are affected, and generally the papillitis is gross, with considerable swelling. In very rare instances one nerve only is involved, but it is common to find a considerable difference in the degree of papillitis on the two sides. Where this difference is marked, it has been suggested that it may be of service in assisting to localise a tumour of unknown position, by indicating the side of the brain involved. Its actual value, however, is very problematical, for the fact of the one papilla being more affected than the other has no simple relation to the position of an intracranial growth, but is complicated by other anatomical considerations. The clinical evidence appears to show that in cases of tumour of the anterior half of the encephalon the papillitis is more marked on the same side as the growth (*i.e.* left greater papillitis, left-sided growth); while, when the tumour is situated more posteriorly, the nerve on the opposite side is the one more involved.

We also find papillitis in association with *meningitis* (especially traumatic, syphilitic, and tubercular basilar); with *cerebral abscess*; and sometimes with chronic cerebritis without gross lesion.

Injuries to the head may also cause swelling of the papilla, through effusion of blood within the skull or within the optic nerve-sheath.

(*b*) Optic neuritis due to *orbital disease* is nearly always one-sided, and of the retro-ocular kind.

The optic nerve may be attacked during an *orbital cellulitis* arising from local injury, or by the extension of erysipelatous or other inflammation from adjacent parts. Sometimes an orbital cellulitis seems to follow exposure to cold. Occasionally a very severe form of cellulitis is associated with a general septicæmia, possibly through thrombosis of the ophthalmic veins.

Orbital periostitis accompanying local bone disease, or spreading from the upper jaw or nasal cavities, is liable to affect the optic nerve, probably by invading its outer sheath, where this and the periorbita are continuous at the optic foramen. To this class, doubtless, belong cases of alveolar abscess, where complaint has been made of tenderness extending up the cheek and into the orbit, with visual failure in this eye, and sometimes accompanied by slight prominence of the globe, and pain on pressure over it. Such an inflammation may readily gain the orbit by the spheno-maxillary fissure.

Orbital tumours, especially when situated near the nerve, may cause optic neuritis; but

more usually they simply give rise to atrophy of the nerve by their pressure.

(*c*) In its passage through the *optic canal* the nerve is exposed not only to inflammation extending to it from the bony walls and the periosteum, but also from the *sphenoidal sinus*, only separated from the canal by a thin lamina of bone, sometimes imperfect. It is probable that many of the cases of retro-ocular neuritis traced to exposure to cold are examples of inflammation attacking the nerve in this part of its course, and sometimes in this way (through the sphenoidal sinus). In such cases both nerves are often affected, and almost simultaneously. Such cases of neuritis are not usually associated with early changes at the optic disc, but later there may be slight or moderate papillitis. The periosteum in this locality seems to be not infrequently the seat of gummatous deposit.

(*d*) Mention must also be made of the alleged occurrence of optic neuritis in connection with disease of the bones of the ear, without the usual intervention of meningitis or of cerebral abscess.

(*e*) The optic nerve itself may be the seat of a local manifestation of a general affection, as a syphilitic gumma or a tuberculous deposit. Again, the sheath of the nerve, possibly in any part of its course, but apparently most commonly near its junction with the sclerotic, is liable to be affected occasionally by inflammation of a gouty or rheumatic character; in many cases of this nature a marked tendency to recurrence has been observed. Papillitis is sometimes caused by an extension of inflammation from a neighbouring ocular tissue (*e.g.* gumma of the adjacent choroid), and it has also been observed in an early stage of sympathetic inflammation before the occurrence of irido-cyclitis.

Here also we may mention the not infrequent occurrence of retro-ocular neuritis in connection with insular sclerosis. In some of these cases the optic-nerve attack is like an ordinary acute retro-ocular neuritis, this nerve being the seat of a local lesion such as occurs disseminated elsewhere in the central nervous system.

Infrequently papillitis occurs in cases of myelitis, both chronic and acute. It is probable, as has been suggested by Gowers, that in such cases the optic neuritis and myelitis are independent, and both due to a common cause.

(2) *Causes of General Influence.*—Occasionally we get papillitis occurring alone in *renal disease*, but usually in such cases there is retinitis in combination with it. In *chlorosis*, or in progressive *pernicious anæmia*, we may meet with papillitis or papillo-retinitis, and also, more rarely, in *leucocythæmia*. In the *anæmia following* repeated large *loss of blood*, as from hæmatemesis, for example, papillitis has been observed, but more commonly we get retro-ocular neuritis or simple atrophy in this condition. Papillitis occasionally occurs in association with menstrual disturbances, such as sudden suppression or habitual irregularities.

Papillitis or retro-ocular neuritis is sometimes found in patients during or recovering from *acute febrile diseases* (typhus, scarlatina, measles, variola, enteric), in *malarial fever*, and also occasionally after *influenza*. In *diabetes mellitus* we may get loss of central vision of a similar character to what occurs in chronic poisoning by tobacco (also from lead, alcohol, and bisulphide of carbon). In as far as the macular fibres are alone or mainly involved in these cases, the visual failure has been ascribed to retro-ocular "axial" neuritis; but doubt has recently been thrown on this view, and the affection of the axial fibres is probably to be regarded as secondary to a poisoning of the retinal macular ganglion cells (*see* art. "Retina"). In some acute instances of poisoning by these agents, however, slight papillitis has been observed.

In certain families we have an hereditary predisposition to suffer from a retro-ocular neuritis of a slowly progressive form, the nerve changes usually occurring about the age of twenty, and almost exclusively in males.

In most of these instances (*e.g.* anæmia, renal disease, fevers, etc.) the general influence causing optic neuritis is doubtless traceable to the presence of some poisonous material present in the blood, which has either a *direct* effect on the ganglion cells or their processes, or an *indirect* effect on these structures through its action on the nutrient blood-vessels.

Prognosis and Treatment.—The prognosis in all cases of optic neuritis must be a guarded one in reference to the final condition of the vision. The ophthalmoscopic appearances presented are of less service to us in this respect than a knowledge of the cause of the affection. We may have a papillitis with good vision leading ultimately to complete atrophy and blindness; while, on the other hand, we may have papillitis with the barest perception of light (as in occasional retro-ocular cases) followed by almost perfect recovery of sight.

In papillitis due to intracranial disease the prognosis depends mainly upon the effect of treatment on the exciting cause. In cases of syphilitic origin, accordingly, the outlook as to vision is relatively favourable, if the patient is subjected to an antisyphilitic general treatment before the stage of atrophy of the nerve has commenced. Even after the occurrence of partial failure of vision from this cause, there is often an arrest of the failure under treatment, and sometimes an actual recovery of vision. When due to intracranial tumours (other than gumma), the papillitis may be greatly benefited by the administration of iodide of potassium; and, in rare instances, it has been observed gradually to subside without visual loss even

when the exciting cause was a malignant growth.

Surgical interference has certainly a decided influence in diminishing the prominence of the papilla in such cases, and that independently of there having been any attempt to remove the cerebral growth; the effect seems due merely to the reduction of intracranial pressure. This diminution of the papillary œdema gives the nerve an opportunity of recovery under treatment by iodides, but it is only in a comparatively early stage of papillitis that real benefit to the nerve can accrue in this way.

The value of surgical operation in cases of ear disease, and in all cases where pus is present within the cranium, lies chiefly, of course, in its effect upon the disease itself, and only secondarily in the benefit done to the accompanying optic neuritis.

In orbital disease, likewise, the treatment of the papillitis is determined by the nature of the affection. Pus in the orbit should be evacuated by early incision. In some few instances disease of the sphenoidal sinus has been diagnosed and treated surgically with good result.

Similarly, in papillitis due to general disease, the prognosis and treatment are dependent entirely on the nature of the disease in the particular case. Those cases of neuritis arising from intense anæmia (not pernicious) are very amenable to suitable medication and hygiene.

In retro-ocular neuritis of gouty or rheumatic nature, the treatment should consist in giving a mixture containing salicylate of soda and iodide of potassium, and in protecting the affected eye from cold. The prognosis in these cases is very favourable. Indeed, in most cases of acute retro-ocular neuritis recovery is to be expected, though not always complete; a central colour defect often remains long after the attack, even when visual acuity as tested in the usual way has been restored to normal.

In all cases of optic neuritis absolute rest of the eyes should be enjoined, though it is impossible to insist strongly upon it when the inflammation lasts (as it sometimes does) for many months. Exposure to bright light must be avoided by the wearing of dark neutral tint glasses.

Atrophy of the Optic Nerve.—By the term "optic atrophy," when clinically employed, it will be understood that atrophy visible at the optic disc is meant. Pathologically considered, the optic nerve is always ultimately affected throughout its entire length in every form of atrophy, at whatever part of its course it may be first involved. Such an atrophy may either be *complete*, affecting the entire thickness of the nerve; or *partial*, when a smaller or larger number of the fibres escape; even in partial atrophy, however, the affected fibres are finally atrophic throughout their whole length.

Etiology.—Cases of optic atrophy are often divided broadly into two great classes, *inflammatory* and *non-inflammatory* or *simple*. Such a division is certainly most useful, but it groups so large a number of different forms of atrophy under a single heading that it is not of much etiological value.

We offer the following account of the different agencies that may produce optic atrophy, in the hope that it may prove serviceable in clinical classification:—

1. From inflammation of the nerve (consecutive atrophy).

(*a*) Papillitis.

(*b*) Retro-ocular neuritis.

2. From interruption in the continuity of the nerve-fibres in any part of their course.

(*a*) Section or rupture, as by wounds or other injuries.

(*b*) Destruction by inflammation, etc.

From the point of interruption atrophy extends along the involved fibres in both directions—peripherally and centrally.

3. From pressure.

(*a*) On the optic disc (glaucoma).

(*b*) On the nerve in the orbit (hæmorrhage into sheath).

(*c*) On the nerve in the optic canal (bony growths or fracture).

(*d*) On the intracranial optic nerve or chiasma (aneurysm, distended third ventricle, tumour of pituitary body).

4. From interference with blood-supply (*e.g.* in peri-papillitic choroidal atrophy).

5. Degeneration of fibres secondary to destruction, or impaired nutrition of ganglion cells.

(*a*) In atrophy of the retina (following total embolism or thrombosis of arteria centralis; in pigmentary degeneration of the retina, primary or secondary; in retinal detachment).

(*b*) In some cases of poisoning (as, *e.g.*, tobacco and bisulphide of carbon; the macular cells and fibres are chiefly affected).

(*c*) From an inherited neuropathy. Possibly the family cases known as Leber's atrophy may come under this heading.

6. Simple degeneration or true primary atrophy. This form frequently occurs in association with disease of the central nervous system, especially with tabes, but occasionally also with cerebral atrophy, mania, general paralysis of the insane, congenital idiocy, and primary lateral sclerosis of the cord. When found uncomplicated with other nerve lesion, the remote causes assigned are the same as those which are given for the occurrence of grey degeneration in the central nervous system; and it would therefore seem probable that, due to the action of certain causes, grey degeneration may occur independently in the brain, spinal cord, or optic nerves, and may be confined to one or other of these situations. Accordingly, we find male adults of the neuropathic dis-

position, from thirty to fifty, predisposed to this form of atrophy. The more immediate causes mentioned are severe bodily fatigue and hardships, especially exposure to cold and insufficient nourishment, grief and anxiety, mental excitement and exhausting brain-work, irregular habits of life, sexual excess, etc. But the great predisposing cause, common to a large proportion, at least, of these cases of grey degeneration, is syphilis. The effect is attributed to the action of a toxine developed in the post-tertiary stage of this disease.

Pathology.—The nervous elements of the trunk of the nerve are involved in all cases of optic atrophy, and, according to the number of fibres affected, there is a corresponding loss of function. The histological condition of the atrophied nerve varies somewhat, according to the nature of the initial pathological process, and dissimilar appearances may be presented in sections through different parts of the same nerve-trunk. Thus, in *atrophy following a neuritis*, we find the usual post-inflammatory changes at the seat of the inflammation, while the remainder of the nerve may present the appearance found after a simple or non-inflammatory atrophic process. As previously mentioned, neuritis leads to hypertrophy of the interstitial connective tissue; the constant contraction of the meshes in which the nerve-fibre bundles lie, subjects the latter to pressure, interfering at first with their function, and ultimately with their structural integrity. The nerve-fibres involved either finally break down and are removed, or they undergo grey degeneration; in the former case the diameter of the nerve is much reduced. In the case of a neuritis at or near the intraocular end of the nerve, the walls of the central blood-vessels are thickened and their lumen is diminished—partly in consequence of this change in their walls, partly from the pressure exerted by the hypertrophied connective tissue surrounding them.

In *simple atrophy* there is seldom much increase of connective tissue. The nerve-fibres lose their medullary sheath by a process of granular fatty degeneration, while the axis-cylinders are usually retained, but converted into fine indistinct fibrils. There is, consequently, little change in the size of the nerve, which has the appearance of a translucent, grey, gelatinous-looking cord. This "grey degeneration," as it is called, is the most common form of simple atrophy. Occasionally we do find considerable thickening of the connective-tissue framework, which may presumably be due, either to a primary sclerosis of a low chronic inflammatory nature, or to a hyperplasia of connective tissue secondary to degeneration and shrinking of the nerve-fibres.

In the great majority of cases both nerves are affected, though one may be considerably in advance of the other. Unilateral optic atrophy is to be explained by the one-sided nature of the preceding inflammation or other local cause.

Symptoms.—The prominent symptom in every case of optic atrophy is failure of vision. The occurrence of this failure is usually gradual, but varies greatly in rapidity in different forms and different cases. There is also much variation in the manner in which the field is affected. An examination of the central vision by test-types is necessarily of great practical importance, but the character of the loss—as regards the visual field, colour-perception, and light-sense—is often of greater value in judging of the causation and in forming a prognosis (*see* articles "Amblyopia," "Vision, Field of").

The nature of the visual failure in cases of atrophy secondary to neuritis has already been described. In atrophy from chronic retinal degeneration, the failure is usually gradual, central vision often remaining acute after the rest of the field has been lost.

In atrophy due to pressure upon the nerve-fibres behind the eyeball, vision often fails suddenly. In other cases the failure is gradual and partial, and sometimes only the temporal half of the visual field is lost in each eye (as in pressure on the posterior part of the chiasma from tumour of the pituitary body).

In simple primary atrophy the loss of vision begins insidiously, and proceeds slowly and continuously, but may, in exceptional cases, come to a standstill while moderately useful vision still remains. Besides the central failure, there is marked contraction of the visual field, sometimes occurring in a uniform concentric manner, but more frequently in a less regular way so as to cause sector-shaped defects. Affection of the colour-sense is almost constant, green being the colour first confused, while later perception fails for red, blue, and, lastly, yellow. In the final stage of atrophy all colour distinctions are lost; but there is no constant relation between the degree of the loss of colour-sense and the amount of the amblyopia.

The condition of the *pupils* at rest should be noted in all cases. They are usually wide in atrophy following neuritis, small in tabetic cases, and either wide or of medium size in other forms. In tabes and in general paralysis the pupils are often unequal in size. If any vision remains, the behaviour of the pupils to light is of importance; in tabes they are inactive, while still contracting well when the eyes converge in looking at a near object (Argyll-Robertson pupil). In other forms of atrophy, so long as vision remains, the pupil continues to show a response to light (*see* article "Pupil").

Ophthalmoscopic Appearances.—The elements of the optic disc which undergo the atrophic change are the nerve-fibres and the capillaries. Atrophy of the capillaries causes pallor, while the nerve-fibre atrophy is ophthalmoscopically

recognisable only when an atrophic excavation exists. This latter, however, is often absent, since the space formerly occupied by the intact nerve-fibres may be filled, or more than filled, by new connective tissue.

We judge of the optic atrophy chiefly, therefore, by the *colour*, but caution must be exercised in the observation, as the disc varies considerably in this respect in different individuals within physiological limits. It is particularly unsafe to diagnose pathological pallor of part of the disc unless the vision be correspondingly affected.

The *excavation* in atrophy comes quite up to the margin of the disc all round, and is recognisable by a slight change in the level of the vessels, and sometimes by their altered course at the edge of the disc. The atrophic excavation is never very deep, and slopes gradually backwards from the margin, so that the condition may fitly be termed "saucering," in contradistinction to the deep, steep-sided "cupping" found in chronic glaucoma (*see* article "Glaucoma"). The occurrence and depth of an atrophic excavation are determined—(*a*) by the size and form of a formerly existing physiological pit; (*b*) by the degree of loss of nerve substance; (*c*) by the amount of new connective tissue formed, and by the contraction which has taken place in this latter. At the bottom of the "saucer" the lamina cribrosa may be visible over a large area, or it may be quite concealed by the newly formed tissue. The central retinal blood-vessels are in some cases much diminished in size; in other cases only very slightly, if at all.

In the form of atrophy occurring after *papillitis* the disc is usually white; its margin is visible, but often ragged-looking from irregular distribution of pigment in the immediately surrounding retina; the central vessels are frequently bordered by opaque white lines, and are considerably reduced in size (particularly the arteries); the excavation is absent or slight, and the lamina cribrosa is invisible.

In what is known as *simple atrophy* the disc has a different appearance. It is markedly pale, but seldom so white as in the last form, while the lamina cribrosa is conspicuous over a great part of its surface; the margin is clear and very distinct, without surrounding pigmentary disturbance; the central vessels are normal in size and character or slightly reduced in size; the atrophic excavation is marked. This condition follows in all cases in which the disc becomes atrophied secondarily to an affection of the nerve quite behind the eyeball—as from gross retro-ocular neuritis, wound, or pressure. More rarely this form of simple atrophy succeeds a papillitis, probably indicating that the actual atrophic process in the disc was not produced by the severity of the changes there, but occurred secondarily from an inflammation that had its greatest intensity behind the globe. In simple degeneration the disc-changes are also usually of this character; not uncommonly in tabes, however, there is less distinctness of the lamina cribrosa than above described.

In the atrophy consequent on choroido-retinal changes, the disc has generally a peculiar opaque, pale, yellowish-pink, "waxy" appearance, and the retinal vessels are greatly diminished in size and sometimes in number.

In the partial atrophy produced by milder forms of retro-ocular neuritis, and in those due to the action of poisons (*e.g.* tobacco), the pallor is often confined to the temporal half or third of the disc, and this appearance frequently persists after recovery of function.

Prognosis.—The visual prognosis is generally unfavourable in all forms of true optic atrophy. In exceptional cases of atrophy following papillitis, considerable improvement takes place, when the sclerosing process does not lead to excessive pressure on such nerve-fibres as have escaped destruction by the inflammation. In some examples of retro-ocular neuritis followed by an atrophied appearance of the disc, there is a wonderfully complete restoration of vision; in such cases the atrophy has mainly or entirely attacked the capillaries on the disc, the nerve-fibres having escaped permanent damage.

In primary atrophy, almost complete blindness usually occurs in from one to three years; but in some instances the atrophic process stops while moderately useful vision still remains. If, along with a slight progressive contraction of visual field, there is a rapid contraction of the field for colour, the prognosis is unfavourable. The presence of relatively good vision with advanced ophthalmoscopic signs of atrophy is a favourable sign.

Treatment.—Optic nerve-fibres when once destroyed are never restored, and the function formerly performed by such fibres cannot be regained. All that can be expected from treatment, therefore, is the preservation of fibres that, though threatened, are as yet intact; this preservation may be permanent or only temporary—a mere postponement or retardation of the destructive process.

In atrophy due to a neuritis the treatment is directed to the inflammatory process, and has already been considered.

In other cases our first endeavour must be to discover the cause. When the atrophy is dependent on some recognised general condition or toxic influence, the treatment must be regulated accordingly. In all forms of atrophy it is most important to prevent, as far as possible, all depressing influences on mind and body. Good food, healthy exercise, and cheerful surroundings will constitute valuable aids to any other remedial measures. Subcutaneous injections of strychnine are worthy of a trial. Absolute rest of the eyes, except in inflammatory forms, is unnecessary. Moderate use is, if not

actually beneficial, certainly not apparently hurtful; and if the atrophy is progressive, and vision doomed to fail in any case, it is surely cruel to prematurely deprive the patient of all use of whatever vision remains. Appropriate glasses should be given to correct any error of refraction, and sometimes to magnify near objects. There must be strict limitation, however, of all finer work, such as reading; a sense of fatigue, or increased difficulty in seeing smaller objects, should be regarded as an evidence of the need for a temporary rest. Galvanism has been tried, but with doubtful benefit; though it is not, we believe, actually hurtful (as has been asserted), its use cannot be recommended.

Injuries to the Optic Nerve.—The optic nerve may be injured in any part of its course, either as the result of a blow, stab, gunshot wound, or fracture of the sphenoid bone. A stab or thrust into the orbit may (without injury to the eyeball) cut or tear the nerve; or may merely bruise it, or cause an extravasation of blood within its sheath; or again, without actually injuring the nerve, may sever the central vessels outside its trunk. If a foreign body penetrate deeply it may fracture the orbital wall near the apex, and thus secondarily lead to injury of the nerve by a displaced bony fragment. Similarly, gunshot wounds may implicate the nerve either in its orbital or intracranial course. Sometimes the intraocular end of the nerve is injured by a fragment of metal that has penetrated the eyeball. Fracture of the base of the skull, as from a fall or severe blow on the head, sometimes causes injury to the nerve at the optic foramen, or behind it.

Sudden complete blindness of the corresponding eye is usually the immediate result of any severe injury to the optic nerve. If the solution of continuity be complete, permanent blindness is the consequence; if incomplete, there may be restoration of vision in part of the field. When the nerve is simply bruised, or pressed upon by extravasation within the sheath, sight may be restored; usually, however, secondary changes afterwards occur, leading ultimately to complete or partial atrophy.

The *ophthalmoscopic appearances* are usually at first of a normal character, but as the atrophic process reaches the disc, it gradually assumes the appearance of an ordinary simple atrophy. If, however, the central artery be cut or torn across, we at once find changes in the fundus similar to those met with in embolism of this vessel.

The *treatment* of the recent injury must be based on general surgical principles. Later, if the continuity of the nerve has been preserved, the measures recommended above in optic atrophy may be tried.

Tumours of the Optic Nerve.—The optic nerve trunk (including its pial sheath) may be the original seat of a tumour. It may also be involved secondarily by the extension of growths arising (*a*) within the eyeball (from retina and choroid); (*b*) in its dural sheath; (*c*) or in the surrounding orbital tissue.

When the nerve is primarily involved by a new growth, there is little or no pain, and the movements of the globe are usually good; but there is early failure of vision, papillitis or optic atrophy, and later proptosis. The most common form of such a tumour is myxo-sarcoma.

Of intraocular growths secondarily affecting the nerve, we must mention retinal glioma and sarcoma of the choroid: the former extends in the medullary substance of the nerve, the latter spreads along the sheath and fibrous framework.

At the optic disc there is occasionally seen a projection of small, pearl-like bodies, of the nature of the "corpora amylacea" found elsewhere in the central nervous system, and not necessarily connected with any visual loss. Gummata seem particularly apt to involve the optic nerve fibres in the optic canal and at the chiasma; in the latter situation, also, deposits of cheesy, tuberculous material are sometimes found.

Congenital Abnormalities.—Imperfect closure of the cleft in the embryonic optic cup and pedicle leads to coloboma of the iris, choroid, or optic nerve-sheath, according to the part and amount involved. This latter—coloboma of the sheath of the optic nerve—is comparatively rare. When it occurs, the main branches of the central vessels are seen to emerge near the upper edge of the disc-area, while the lower part of the latter, corresponding to the cleft in the sheath, is very pale and deeply excavated, with small vessels curving round its margins. The appearances vary considerably in different cases, and the condition is generally accompanied by coloboma of the neighbouring part of the choroid below.

The Retina

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Anatomy.—The retina is to be regarded as a distinct offshoot from the central nervous

system, in which structural adaptations have occurred necessary for its special function of being stimulated by waves of light. The stimuli so produced—which vary in character according to the length and height of the waves—it is also capable of transmitting to the brain, by means of the elongated processes of its ganglion cells, familiarly known as optic-nerve fibres.

It will be sufficient for our present purposes to describe the anatomy of the retina very briefly. It consists of two distinct parts—*pigment-epithelium* and *retina proper*. The former lies immediately on and is closely attached to the inner surface of the choroid, and is formed by a single layer of large pigmented cells; in these cells subtle changes, probably mainly chemical in nature, are produced under the influence of light. The retina proper lines the greater part of the interior of the eyeball, beginning a little behind the ciliary body and reaching to the optic disc: it is made up of several layers—nerve-epithelium, nerve-cells, and nerve-fibres—held together by a supporting framework of delicate connective tissue. The nerve-epithelium is in contact with and slightly embedded in the pigment-epithelium; the absence of any true structural union between these two epithelial layers renders a separation liable to occur under certain circumstances. On the inner surface of the retina are the nerve-fibres, which converge and meet at the disc. Between these surface layers are numerous nerve-cells and their processes. The most important of these next the fibre layers are large ganglion cells, whose greatly elongated central processes (axons) constitute the fibres just mentioned in the retina, and then run and are continued by the optic nerves and tracts to gain the basal ganglia of the brain. The larger blood-vessels of the retina lie near its inner surface, while the smaller branches run both in the inner and middle retinal layers, but none enter the nerve epithelium. The arteries do not anastomose with one another, nor is there any communication between these vessels and those of neighbouring structures after leaving the disc, so that the retinal circulation is terminal. Near the centre of the retina (to the outer side of the disc) is a small spot called the *fovea centralis*, where the epithelial layers (pigment and nerve epithelium) are alone present.

Ophthalmoscopic Appearances.—On ophthalmoscopic examination, the normal retina, apart from its blood-vessels, is practically invisible, as it does not manifestly interfere with the passage of light to and from the chorio-capillaris. It is not *quite* transparent, however, and we can usually detect the presence of the retinal pigment as a very fine, dark stippling, visible on the red fundus when the pigment is very well developed. In persons of dark complexion, and particularly in dark races, the red fundus reflex is very decidedly obscured; while this reflex is particularly bright in light-complexioned people, on account of the small amount of pigment present. About two disc-diameters to the temporal side of the disc, the "yellow spot" region is recognisable by its darker shade of red, destitute of visible blood-vessels; near the centre of this area is a small brighter spot, marking the position of the fovea centralis. The retinal blood-vessels should be carefully studied in normal eyes, particularly in young people. We should note the relative colour and size, and the character of the central light-streak in the arteries and veins. It will be observed that both sets of vessels usually branch dichotomously.

Diseases of the retina can only be detected by ophthalmoscopic examination. As already mentioned, the retina, with the exception of its blood-vessels and its pigment-epithelium, is almost quite transparent, and is therefore practically invisible ophthalmoscopically. Its diseased conditions are recognised, accordingly, by *changes in its blood-vessels* or *in its pigment-epithelium*, or by a *loss of its normal transparency*. The loss of transparency may be general or localised, sharply limited or diffuse, and such opacities may vary greatly in shape, size, and colour; we may mention as examples, retinal œdema, hæmorrhages, and inflammatory exudations.

Changes in Retinal Blood-Vessels.—Disturbances in the retinal circulation may be shown by changes in the size and course of the vessels, in the appearance of their walls, and by a visible pulsation. In estimating the *size* of the retinal vessels we regard chiefly the larger branches on and near the optic disc, comparing them with our mental impression of those in an average normal eye. The departure from normal may apply to all the vessels, or to the arteries or veins alone; or it may only be observed in one or two branches; or again, the same vessel may show an irregularity in its calibre at different points. On making observations on the relative size of the arteries and veins, it is to be remembered that the normal artery has a diameter of about two-thirds that of the corresponding vein. By the direct method of observation the vessels will appear larger in the myopic and somewhat smaller in the hypermetropic eye, while by the indirect method an opposite effect will be produced. An erroneous estimation of size may also be caused by failing to note an abnormal rate of subdivision—the earlier the branching occurs the smaller the individual vessels will be at a given distance from the centre of the disc.

(1) A *general enlargement* of the retinal vessels, with an unusually dark blood-column, is present in congenital defects of the heart with cyanosis. The retinal vessels are also found somewhat distended in extreme cases of Graves' disease. A *general reduction* in the size of the vessels is occasionally present in marked anæmia

and in renal disease; it is particularly conspicuous in cases of chronic retinal atrophy.

(2) The *arteries alone may be small* in cases of optic neuritis (late stages), of embolism, arterial thrombosis, and of quinine poisoning; occasionally also in general anæmia (*e.g.* after great hæmorrhage), where the veins appear normal, or even large from being flattened, owing to their reduced intravascular tension. Rarely, the arteries are temporarily reduced in size from spasm of their walls.

(3) In *venous distension* these vessels are large, rounded, abnormally dark, and tortuous. This condition may be dependent on local or general interference with the venous blood-flow. When the cause is local the distension is sometimes very great, and hæmorrhages are common. Among local causes may be mentioned optic neuritis, venous thrombosis, and pressure exerted by a diseased overlying retinal artery, as the most common. The retinal veins may be distended as part of a general venous congestion in the head, from pressure on the great veins of the neck. Again, the retinal congestion may be part of a general venous distension, either temporarily, as from coughing or straining at stool; or continuously, as in mitral disease and pulmonary emphysema.

(4) Occasionally we find irregularity in the size of both arteries and veins in different parts of their course over the retina. For example, an arterial branch of normal size near the disc may soon quickly dwindle without having given off any branch meanwhile; on tracing it farther out, this vessel, or one of its subdivisions, again swells out to its normal calibre, or even larger. In the same fundus the veins will usually also exhibit marked irregularities in size where crossed over by arteries, an interruption seemingly taking place at this point, and the vein being distended on the peripheral side of the crossing. In these cases there is hyaline degeneration with enlargement of the outer and middle arterial coats, and the pressure thus produced on the underlying thin-walled vein interferes with its blood-flow. The appearances described are usually associated with granular kidney (see later).

Increased tortuosity is more common in the veins than in the arteries of the retina. Such a course is always found in congested retinal veins. When in the plane of the retina the exaggerated tortuosity is easily observed; an antero-posterior curve also occurs, and is recognised by the vein being there darker than elsewhere, and by a local absence of the central light-streak.

The arteries are usually unduly tortuous when affected by the hyaline degeneration mentioned above.

In exceptional cases, normally sized vessels, both arteries and veins, show great tortuosity; this condition has been observed in connection with a liability to severe headaches in some hypermetropic persons.

Changes in the vascular walls are often recognisable with the ophthalmoscope:—

(1) A comparatively common form presents the appearance of distinct narrow white lines bounding each side of the red blood column. It is more frequent in the arteries than in the veins, and is then due to thickening of the adventitia, or to a deposit of white cells around the vessel. The perivascular lymph space of the veins may be the seat of a similar deposit. This vascular change often follows inflammation of the retina, as, for example, in the form accompanying papillitis, and is best seen on the larger vessels at and near the disc. But the same appearance may occur independently of any inflammation. Occasionally the entire breadth of the artery looks white, or there may be merely a narrow red line in the centre; yet in such a case there is no necessary loss of vision, since the lumen of the vessel may not be involved.

(2) In some cases the arteries have a much brighter reflex than usual, with a brilliant central light-streak. This appearance is usually accompanied by the other phenomena mentioned as characteristic of hyaline degeneration.

(3) Aneurysms may occur either on the larger branches of the central artery, or on the smaller twigs and capillaries of the retina. The latter are of clinical importance from their having been found associated with a similar condition of the small vessels in the brain.

Visible pulsation in one or more of the large *veins* on the disc is not uncommon in normal eyes; it is best seen just at the edge of the physiological pit. It also occurs, pathologically, from the same causes that produce *arterial pulsation*. This latter is never normally present, but occurs in aortic regurgitation, and in increased intraocular tension, whether from disease, or mechanically produced, as by finger-pressure from without. It has also been observed in acute anæmia from profuse hæmorrhage. Arterial pulsation is usually visible only in the large vessels on the disc, but in aortic cases it may sometimes be traced peripherally in branches of comparatively small size.

A peculiar form of visible circulation in both arteries and veins is seen not infrequently in recent embolism of the arteria centralis (see later).

Abnormalities in the Pigment of the Retina.—A congenital deficiency of this pigment is general over the entire fundus, and is familiar to us in partial or complete albinism. Pathological changes also frequently take place in this layer. Over a greater or less fundus area the pigment may be removed from its normal situation, and we thereupon get an unusual reflex, differing in character according to

the degree in which the choroidal tissues have participated in the pathological change. Pigment so disturbed may be visible as a diffuse, coarse peppering over the red fundus-reflex, or it may be collected in larger masses of various forms over the denuded area, or again aggregated in the form of a fairly regular ring surrounding the affected patch. Such pigmentary changes are usually rightly considered along with their associated choroidal diseases, but one particular form (retinitis pigmentosa) will receive our attention in this article when we come to consider atrophic changes in the retina.

Occasionally, in persons beyond middle life, we see numerous round, yellowish-white, translucent spots of small size, bordered with pigment. They may be found in any part of the fundus, but are particularly common in the macular region and in the extreme periphery. They are due to the presence of small colloid bodies projecting into the retina, probably derived from the cells of the pigment-epithelium, and associated with an altered nutrition of this layer. When near the fovea they cause some disturbance of vision, but are not otherwise of clinical importance.

Apart from circulatory and pigmentary changes, we may recognise retinal affections by an interference with the normal transparency of the retina. Thus we may have diffuse haze from œdema, red patches from hæmorrhages, black deposits of pigment, or opaque white areas from exudation or from fatty changes. Again, the retina may be visibly raised from its normal position in the fundus as the result of an exudation or solid growth.

Hæmorrhages may occur in the retina at any part of the fundus, and are readily recognised, when recent, from their bright red colour. There may be only one, or they may be practically countless; and they may vary in size from a just visible spot or streak to a patch much larger than the optic disc. They are most common near the large vessels in the nerve-fibre layer, and then have a striated or flame-shaped appearance from the extravasated blood having passed between and run along the nerve-fibre bundles. Sometimes they occur from the fine vessels in the inner nuclear layer, and are then usually small and rounded in form. Occasionally a very large, sharply defined hæmorrhage is found covering the macula; in this case the extravasation is more or less rounded, sometimes with a flat upper edge, and is situated between the hyaloid and internal limiting membranes. A large hæmorrhage often bursts through the hyaloid membrane into the vitreous, or sometimes passes backwards and causes detachment of the retina. Hæmorrhages interfere with sight according to their size and position; those mentioned as occurring at the macula necessarily cause loss of central vision, though in this case the prognosis is good. Very commonly their presence is of less visual importance, in so far as sight has been already impaired by the disease of the optic nerve or retina, of which the hæmorrhages are merely a result. Absorption takes place gradually, its rapidity being greatest in hæmorrhages of small size and in those unassociated with disease of the blood-vessels. There is often no trace ultimately left, or there may be a dark pigmentation or brilliant yellowish-white spots marking their former position.

Causes.—Hæmorrhages may be part of an inflammation of the retina or optic nerve, or they may be dependent directly on general conditions, or on retinal disease consequent on general conditions. The extravasation may be due to rupture of vessels or to diapedesis from a vein. The following statement will serve to show the possible manner of their occurrence:—

A. *Rupture of vessels* from—

(1) *Increased intravascular pressure*, local or general. (*a*) *Local*, *e.g.*, from contusion of the eyeball, or from great venous distension, as in venous thrombosis or other manner of interference with venous return. (*b*) *General*, as from cough, or other violent effort, and perhaps from high arterial tension, with or without hypertrophy of the left ventricle.

(2) *Sudden diminution of the vitreous support*, as in wound of the globe.

(3) *Weakness of a degenerated vascular wall*, as in sclerosis and miliary aneurysms of the retinal vessels, sometimes in embolism or thrombosis of the small arteries. Such vascular changes are usually concomitants of some general affection, such as cardiac or renal disease, or diabetes.

B. *Diapedesis* may be the result of—

(1) *Increased Intravenous Pressure.*—This is not always a direct effect; indeed, the retinal veins, on account of their capacity for becoming dilated and stretched, may resist for a long time. But this disturbance and slowing of venous flow leads to an œdema of the corresponding retinal area; and as the walls of the vessels in this area suffer in consequence (along with the other retinal tissues), they undergo degenerative changes, leading finally to diminished resistance to the passage of the red blood-cells through them. Hæmorrhages occurring in this way often remain near the vein from which the blood has escaped, but not invariably so. Not infrequently, when the veins are engorged and the retina œdematous, one or more hæmorrhages are seen lying alongside the corresponding artery. This position, however, is no evidence of the blood being arterial in origin; it is more probable that it passes from the vein slowly along between the nerve-fibres, where there is least resistance, until it meets the obstruction caused by the body of the artery, and then spreads along its side.

(2) *An Altered Condition of the Blood.*—This is possibly the operative cause, in part at least, of the hæmorrhages found in cases of diabetes, severe anæmia, leucocythæmia, ague, purpura, scurvy, and pyæmia.

Retinal hæmorrhages, with tendency to recurrence, are occasionally met with in young people, particularly men, without any obvious cause. These hæmorrhages may be small and peripheral, and of small consequence; in other cases, or later in the same individuals, they are larger in size, often bursting through into the vitreous, and not seldom followed by detached retina and loss of sight.

The *treatment* of retinal hæmorrhage must necessarily vary with the general and local states with which it is associated or on which it depends. In recent cases of large hæmorrhage into the vitreous, iced compresses may be applied over the eyelids.

Inflammation of the retina is usually the result of some general disease, and the classification of its forms commonly adopted is clinical rather than pathological. When an inflammation commences in the retina, the papilla is generally secondarily involved; and, again, retinitis, mainly of the inner layer, is not infrequent in cases of papillitis.

The principal ophthalmoscopic evidences of retinitis are—loss of retinal transparency, dilatation of the veins, and a tendency to the occurrence of hæmorrhages and white patches. Sometimes there are numerous opacities in the vitreous. Anatomically, we find serous infiltration of the retina, inflammatory exudations, hæmorrhages in the inner or middle layers, fatty degenerations, swelling of the Müllerian fibres, and hypertrophy and sclerosis of nerve-fibres.

The impairment of vision, both temporary and permanent, varies much in different cases. The *treatment* must be regulated mainly by the general condition of the patient. Complete rest of the eyes must be enjoined, along with the use of dark, neutral tint glasses. Counter-irritants to the temple are occasionally beneficial.

Hæmorrhagic retinitis is serviceable as a clinical term in cases where we have numerous hæmorrhages along with evidence of retinal inflammation. It usually affects one eye only, and is seldom found before middle life is passed. The hæmorrhages are generally small and numerous, chiefly in the inner layers of the retina; they are sometimes widely distributed over the fundus, at other times confined to the distribution of one retinal vein. The papilla is hyperæmic, with blurred margins. There is diffuse opacity of the retina from œdema, which may be general or localised. All the veins may be very greatly distended, or there may be a less degree of fulness in only one large branch. We frequently find a few circumscribed white spots, and occasionally these assume the stellate distribution at the macula that is so common in renal retinitis.

Causes.—There are two types of this affection indicated in the above description, each probably with a distinct mode of occurrence.

(*a*) When the veins are very greatly distended, while the arteries appear normal, the operative cause is possibly a venous thrombosis. This may affect the vena centralis or one of its branches; in the latter case the venous engorgement, œdema, etc., are limited to the drainage area of the affected vessel. The reason for the occurrence of the thrombosis is generally obscure; there is usually, probably, a surrounding inflammation, implicating the vein secondarily.

(*b*) The veins are not much distended, and look normal generally on the disc itself.

The point of obstruction in the venous flow in such a case is usually ophthalmoscopically visible in one or more branches, and is then evidently due to the pressure exerted by an overlying retinal artery. The arteries present the appearances described above as characteristic of hyaline degeneration, the secondary and tertiary branches of the arteria centralis being particularly bright and "silver-wire" looking. Not infrequently we can, by tracing the obstructed vein towards the periphery, observe œdema and hæmorrhages in its area. The macula is sometimes involved in the œdema; we then usually find a few white spots in this situation, and occasionally these are arranged in rays diverging from the fovea (macular stellate figure); while exceptionally the extreme periphery of the macular neighbourhood is surrounded by a circle of white, irregular in breadth (circinate retinitis).

The œdema, hæmorrhages, white spots, etc., are secondary to the venous obstruction, which again is dependent on the arterial disease. This latter is, in the great majority of cases, if not always, only part of a widely diffused arterial degeneration, which tends particularly to affect the minute arteries of the retina, pia mater, and kidney. Patients with this retinal condition are very often gouty, and they frequently give a history of having suffered from severe migraine. In all such cases the urine should be examined carefully and repeatedly for the presence of albumin; the particular form of renal disease associated with this condition is the granular kidney.

The local prognosis is unfavourable, though sight sometimes improves for a time. The general prognosis is also grave, in as far as there is a marked liability to the occurrence of cerebral thrombosis or hæmorrhage.

Albuminuric retinitis is a term long in general use, to indicate a definite ophthalmoscopic picture of great clinical significance. It is characterised particularly by the presence of numerous soft-looking, light-coloured patches, and of small, opaque, sharply cut, very white

dots or striæ at the macula. The affection of the papillæ varies between a mere slight congestion and a decided engorgement; in the latter case the veins are distended, and there is a marked tendency to hæmorrhages, which occur mainly in the nerve-fibre layer. The soft-edged light patches occur comparatively early, and are found mainly in the neighbourhood of the papilla; they are of rather large size, and usually lie behind the retinal vessels, but sometimes in front of them. The white punctate spots appear later, and are arranged in the form of a fan or star, the lines radiating from the fovea centralis. Both eyes are nearly always affected.

Causes.—This form of retinitis is most commonly associated with chronic disease of the kidney, especially the contracting granular form. It is also sometimes met with in acute nephritis after scarlatina, and is often present in the albuminuria of pregnancy. It must be remembered, however, that we may have a papillitis or papillo-retinitis, very like that from intracranial affections, in renal disease, without the typical appearance above described. On the other hand, retinitis of the albuminuric type, with stellate macular figure, etc., occurs exceptionally in connection with intracranial affections, diabetes, or anæmia, without there being evidence or other suspicion of renal disease. The most valuable factor in the ophthalmoscopic diagnosis in all doubtful cases is the condition of the retinal arteries; if these have undergone hyaline degeneration, renal disease is almost certainly present. This arterial change is, we believe, always present in the retinitis found along with the albuminuria of pregnancy, indicating that such patients are, in fact, the subjects of chronic renal disease, and that pregnancy has had a prejudicial effect on the malady.

Prognosis.—(*a*) Local.—If improvement takes place in the general condition or renal affection, the retinal changes may subside, but the changes at the macula persist for a long time even in the most favourable cases, *e.g.* those occurring after scarlatina and during pregnancy. In the last-mentioned group the prognosis is relatively more favourable if the retinitis has not occurred during any previous pregnancy, and if it occurs during the later months. Induction of premature labour is often advisable —always if the retinal condition is grave or getting rapidly worse.

(*b*) The prognosis as regards life is unfavourable in cases of albuminuric retinitis in chronic renal disease, except where pregnancy has been the determining factor, the subjects of it often dying within a year after the vision has become thus affected.

Syphilitic Retinitis.—This is usually an inflammation of a chronic diffuse type, without gross ophthalmoscopic changes. The retina becomes hazy and grey, especially near the papilla, at the macula, and along the larger vessels. The large veins are somewhat distended, and the outline of the papilla is blurred. White patches and hæmorrhages are rare. The vitreous often shows fine dust-like opacities close in front of the retina. Occasionally, on the inner surface of the retina, one or more white bands of exudation are formed, which afterwards are organised into connective tissue, and may ultimately occasion detachment of the retina. Choroiditis is often an accompaniment. The retinal affection usually comes on late in the secondary stage, often about the end of the first year, and is relatively more common in persons who have acquired syphilis rather late in life; it is also met with, however, in young children who have inherited the disease. It may occur in both eyes, or in one eye only. Later, the retina often shows atrophic changes, with pigmentation.

Course.—There is early visual failure, often with night-blindness, and sometimes micropsia. The retinitis lasts for months, with a tendency to occasional temporary improvement; the ultimate result is usually favourable if proper treatment is pursued. When atrophy and pigmentation result there is much permanent loss of vision.

Treatment.—It is impossible to insist too strongly on the necessity of early mercurial treatment, preferably by inunction. The *local* treatment of retinitis has already been described.

Leucocythæmic Retinitis.—Retinal changes not infrequently occur in the splenic form of leucocythæmia, and are then always present in both eyes, though often in an unequal degree. In a typical case of this affection there are retinal hæmorrhages of peculiar character, appearing as white patches surrounded by a red border; these are found particularly at the limits of the retinal circulation, viz. at the macula and at the periphery of the fundus. There are also ordinary hæmorrhages, usually of small size, and widespread retinal haze with blurring of the disc. The general fundus-reflex is of an orange-yellow colour; the retinal arteries are bright orange, and the veins broad, flattened, and of a light carmine tint.

Diabetic Retinitis.—Retinitis is not common in diabetes, but when it occurs is nearly always found in both eyes. There is seldom haze of the retina or change in the papilla. The most common form of this affection shows at first simply many small retinal hæmorrhages; but in some cases we have later rather large, flat-looking, white, opaque spots at the macula, with sharp, crenated borders. In other cases the appearances closely resemble those found in renal disease, but in these cases the arteries show hyaline degeneration with pressure on the veins, and granular kidney is probably present in addition.

Central retinitis from exposure to direct sunlight has been occasionally met with in persons who have looked at an eclipse of the sun without adequate protection. Immediately after the exposure there is some loss of central vision, in the form of a central scotoma. In very severe cases this remains permanent, but generally improvement slowly takes place. Ophthalmoscopically the macula is found to be more darkly pigmented than usual, with some large granular deposits near the fovea, which latter is visible as a greyish yellow spot. In severe cases with permanent scotoma there is probably some destruction of the outer segments of the cones, as well as change in the pigment epithelium.

The *treatment* should consist mainly in the use of dark glasses. Leeches or counter-irritants applied to the temple have been recommended.

Central Retinal Change in Infants.—Mention must be made of this rare affection, which has important clinical and pathological relations. About the age of three or four months the child is noticed to be listless, with weakness of the muscles and apparently imperfect sight. The ophthalmoscope shows a dense grey, slightly raised opacity occupying the macular region, in the centre of which is a bright red spot—a condition like that found in embolism of the central artery. The disc may even now be pale, and becomes progressively more atrophic. Soon the child becomes quite blind, with pupils inactive to light; there is an apathetic state and partial muscular paralysis; finally, nourishment fails and death takes place about the age of two years. The ophthalmoscopic changes are always present in both eyes. The disease sometimes affects several members of a family. Pathologically, atrophic changes have been found in the neurones of the cerebral cortex, and presumably similar changes occur in those of the retina. No treatment is of any avail.

Central Retinal Affection from Toxic Influence.—The particular form of visual failure known as *toxic amblyopia* has generally been considered under the diseases of the optic nerve, as the recognised changes in the fibres seemed to indicate that the affection was a variety of retro-ocular neuritis. Recent knowledge regarding the constitution of the neurone has rendered necessary a reconsideration of the hitherto accepted pathological nature of this particular affection of the nerve fibres, and there is good reason to believe that the large macular ganglion cells in the retina first suffer under certain toxic influences, and that the implication of their *axons* or nerve fibres is in reality secondary. There is no certain knowledge as to the exact manner in which the ganglion cells are thus affected—whether it be a direct poisonous influence communicated to them through the circulation, or whether the nutrient arteries are themselves first damaged by the poison conveyed in the blood.

Tobacco is by far the most common toxic agent in this disease, and the subjects are nearly all males at or beyond middle life. As a rule, these persons have used strong, coarse tobacco, and have either smoked or chewed (sometimes both) a comparatively large amount. In a considerable proportion of the cases there is a history of recent mental shock or worry, which probably predisposes the nervous system to suffer, but which also frequently induces a larger consumption of the drug. Other toxic agents, that either initiate similar effects or predispose the nerve cells to suffer from tobacco, are lead, alcohol, and bisulphide of carbon. Diabetes, and perhaps also granular kidney, have a similar influence.

The visual failure is progressive, occurs in both eyes, and is most marked in bright lights. On examination, the defect is in the central part of the visual field, occupying the fixation area and a little to its outer side, where there is marked colour-blindness for green and red.

Ophthalmoscopic examination commonly shows slight pallor of the outer third of the disc. In some cases a few round, brightish yellow dots of moderate size are observed at the macula, but it is doubtful how far these are associated with the toxic changes, as similar dots are not uncommon in this region in persons with normal vision. In a very considerable number of cases —especially those occurring in oldish men when there is a delayed recovery of vision under treatment—evidences of hyaline degenerations of the retinal arteries are observable.

The *treatment* consists principally in an absolutely strict abstinence from tobacco in every form, and in avoiding any other influence that may be fairly judged detrimental. Good food and fresh air are important, and it is usual to give strychnine in small doses. Recovery of sight rarely commences for several weeks, but then as a rule proceeds rapidly and satisfactorily (*see also* "Amblyopia," vol. i. p. 115).

Retinitis proliferans is a term applied to an affection of the fundus, in which thick, white strands of connective tissue cover part of the retina, encroaching on the vitreous, with vessels of new formation visible over them. The condition is probably always due to the organisation of previous large retinal hæmorrhages.

Retinitis pigmentosa designates a disease in which a certain group of symptoms and a definite course are usually found in association with pigmentary changes in the retina. The term has long been in general use, and has been retained here on that account; but it must not be understood that this affection is inflammatory, and we will find that it may occur without the usual pigmentary changes. The change is properly a chronic progressive atrophy of the retina; it may occur either as a primary

disease, or secondary to choroidal inflammation, and is generally, but not always, associated with peculiar deposits of pigment in the atrophied tissue.

The primary or idiopathic form of the disease is symmetrical and very slowly progressive, usually beginning in early life, and often terminating in blindness about the age of fifty. Night-blindness is the earliest and most characteristic symptom. There is next loss of visual field, generally first in the form of an annular scotoma, which broadens both outwards and inwards until a small central field alone remains. Central vision may remain good for a long time, but gradually deteriorates, and opacities forming at the posterior pole of the lens still further reduce the sight. The affection is often found in several members of the same family, and sometimes in successive generations. Consanguinity of parents is occasionally found, but it is doubtful if this is the direct cause even in these cases; this relationship may simply intensify an already existing predisposition. The true etiology is quite obscure.

Ophthalmoscopic examination shows, nearly always, much pigment in the retina, largely in its inner layers; here black masses, shaped somewhat like bone corpuscles, lie superficially to the level of the retinal blood-vessels. This pigmentary deposit is found chiefly in a belt midway between the centre and periphery of the fundus, and there is also thinning of the pigment layer, with probably atrophy of the chorio-capillaris, so that the deep choroidal vessels become clearly visible. No constant relation exists between the amount of pigment and the loss of sight; indeed, as has been already mentioned, we may have the usual symptoms and course of the disease without the presence of any abnormal pigment in the retina. What is visually of more importance is the extent in which the circulation of the retina suffers, and this varies considerably. In the worst cases there is marked diminution in the size of the retinal vessels, especially the arteries, which may be reduced to mere fine threads; and the disc shows an opaque, yellowish red, waxy atrophy.

Similar changes in the retina and optic nerves are found, not infrequently, secondary to choroidal inflammation; in such cases, of course, we have the atrophic changes in the choroid in addition, and usually of the disseminated type. When the retinal changes occur, primarily or secondarily, the clinical symptoms are the same.

Pathological examination of the retina shows an increase of the connective tissue framework, atrophy of the nervous elements, and thickening of the walls of the arteries. There is atrophy and disintegration of the pigment epithelium, and the retina proper is infiltrated with aggregations of pigment granules, thence derived. These travel forwards in the lymph channels of the retina, particularly those surrounding the retinal vessels, and collect in large conspicuous masses at the vascular branchings.

In a variety of the affection known as *retinitis punctata albescens* there is not only absence of the usual pigmentation, but there are numerous small white spots which are distributed fairly uniformly over the fundus, rather in excess at the periphery.

Treatment.—Galvanism is, we believe, the only form of treatment of any avail in retinitis pigmentosa. It sometimes causes a marked temporary improvement both in field and in central acuity. If it prove serviceable in any case it should be persevered with for some considerable time, and resorted to again when there is a renewed visual failure.

Embolism and thrombosis of the central artery may be considered together, as they cause similar effects on vision, and are accompanied by identical retinal changes. The arteria centralis itself may be implicated, or one of its branches, and the obstruction may be complete or partial.

Embolism is the probable cause when there is cardiac disease, especially mitral, or atheroma of the aorta. It is more common in the left eye than in the right. When Bright's disease is present, and particularly when the retinal arteries show hyaline degeneration, *thrombosis* is to be suspected; not infrequently temporary attacks of loss of vision precede the permanent one. In complete obstruction of the artery, from either cause, the leading symptom is sudden blindness of the affected eye. There is sometimes a temporary improvement a few hours later, from the establishment of an imperfect collateral circulation at the disc, but the usual final result is absolute blindness. In obstruction of a branch the visual defect is generally partial from the first, the final result likewise being loss of vision corresponding to the distribution of the affected branch.

Ophthalmoscopic Appearances.—Within a few hours of the occurrence of obstruction the retina shows a diffuse milky opacity from œdema, best marked in the macula and the neighbourhood of the disc. In the centre of the opaque macula a bright red spot indicates the position of the fovea centralis, where the normal chorio-capillaris red reflex shines through the thin retinal epithelial layers, and appears exceptionally red by contrast with the surrounding cloudiness. The disc is pale, with soft, blurred edges. The arteries are much narrowed, and no pulsation can be evoked by pressure on the globe.

After a day or two the retinal arteries become broader again from partial refilling, and the blood in some cases is seen, both in the arteries and veins, in the form of short, detached columns separated by clear intervals; these columns move in a jerky fashion, with

or against the normal direction of blood-flow. This visible circulation seldom lasts more than a day or so. Small hæmorrhages often occur near the disc.

Some days later the normal arterial pulsation on finger pressure may return in the larger branches, and the retinal œdema slowly disappears, but the nutritive supply of the retina has been too long interrupted for any restoration of function to take place, and the disc gradually becomes secondarily atrophic.

Where a branch only is affected, changes similar to the above are found confined to the area of its distribution.

Treatment is seldom of any avail in embolism, and then only within the first few hours. Our endeavours must be to break up and displace the arterial plug; and massage of the globe has in a few instances proved successful in effecting this. It is probably best performed by alternate, prolonged, moderately firm pressure on the globe and sudden removal of this pressure.

Thrombosis of the central vein has been already discussed when speaking of *hæmorrhagic retinitis*.

Detachment of the Retina.—In this condition the retina proper is separated from the pigment epithelium, and the interval is usually occupied by a serous fluid. The retina is normally very loosely attached, except at the margin of the disc, and at the ora serrata. Fluid once present here readily gravitates to the lower part of the fundus, where detachments of old standing are most commonly found. Occasionally, instead of serous fluid, the displacing agent is an extravasation of blood, or a plastic exudation from choroiditis, or a solid choroidal tumour pushing the retina forward, or a subretinal cysticercus. The last mentioned is very rare in this country. Subretinal fluid may be effused, *primarily*, as a hæmorrhage, or as a serous exudation in connection with inflammation, renal disease, or tumour of the choroid. The common reason of detachment, however, is some alteration in the vitreous—either a mere diminution of its support to the retina, or contraction of connective-tissue formations within it, forcibly dragging and sometimes tearing the retina away from its normal position. In such cases the subretinal effusion of fluid is *secondary*, and either derived by transudation through the choroidal capillaries, or by direct passage through the retinal rent of fluid from the vitreous cavity.

Alterations in the vitreous provocative of detachment are well known to be often associated with myopia; they also occur from inflammation of the ciliary body and choroid. Perforating wounds of the sclerotic may lead to detachment, either immediately, from a loss of vitreous, or subsequently, from contraction of cicatricial tissue at the point of puncture. In many cases of detached retina no cause can be discovered.

Anatomically, the detached retina undergoes a gradual degeneration, the nervous elements being destroyed and its connective tissue hypertrophied.

Diagnosis.—The general appearance of the eye is usually normal. In cases of very extensive detachment the pupil acts imperfectly to light; the anterior chamber is deep, particularly at the base of the iris. The tension of the globe is usually decidedly diminished, but may be normal or even increased; the latter is suspicious of the presence of a choroidal new growth.

Central vision may or may not be much affected, according to the condition of the media and to how far the macula is implicated. The visual field will show a contraction corresponding to the extent of the detachment. But the accurate diagnosis of the disease depends upon the ophthalmoscopic examination. When we illuminate the patient's eye with the ophthalmoscopic mirror, held at about the distance of a foot, we have a want of the normal uniformity of reflex, part of the fundus corresponding to the detachment appearing bluish or light grey, in contrast to the normal red seen elsewhere. This discoloured, detached portion is often folded and more or less tremulous, and the vessels are usually visible, even by this method of examination, appearing as dark, stiff-looking, tortuous lines. In the case of a recent shallow detachment the difference in reflex may not be easily noticeable, but the vessels have the characteristics just mentioned, though they are not so conspicuous, owing to the absence of the strong contrast furnished by the light-coloured surface. By the direct ophthalmoscopic method we now carefully measure the refraction of the vessels on the affected and unaffected parts of the retina, so as to, in difficult cases, finally determine whether a displacement of retina really exists. The vessels show no central light-streak. If detachment is present, estimate the difference of level between it and the normal fundus in its neighbourhood. Having thus satisfied ourselves of the detachment and measured the displacement, we next note its extent, the character of the boundaries, its mobility on movements of the eye, its colour, its variation in height, and the degree of folding. We are thus enabled to form an opinion as to the probable nature of the displacing agent—whether a rounded, solid growth; a low, flat, firm exudation; or a thin fluid. Occasionally, however, the presence of opacities in the media or iritic adhesions interfere with such an examination; under such conditions the indirect method of ophthalmoscopic observation is particularly valuable.

The discoloration of a detachment is due to

opacity of the retina, produced largely by œdema; the darkness of the blood-vessels is accounted for partly by absence of the central light-streak, partly by contrast, but mainly by their being seen by transmitted light.

Symptoms and Cause. — There is often a history of a comparatively rapid impairment of vision; objects sometimes appearing distorted or as covered by a thick black veil. Sometimes we have complaints of quick flashes of light. As already mentioned, central vision may remain good for some time, but in a large number of cases the detachment gradually extends, and the sight becomes progressively worse, until ultimately there is complete blindness or only the barest perception of light. The lens often becomes opaque in the case of large detachments that have lasted for some time.

Treatment.—In recent cases of serous detachment we can often get satisfactory improvement of vision, for a time at least. But the necessary remedial measures are decidedly irksome, and even trying to the general health, if all the indications are strictly followed out. What we desire is absorption of the subretinal effusion. Rest, lying on the back, in a dimly lighted room is particularly to be insisted on for several weeks, and the eye should be kept covered with a moderately firm pressure bandage. All exertion or sudden effort must be avoided, and the bowels carefully regulated. Diaphoretics are recommended, and even purgatives, but such a course is necessarily very lowering, and the patient's general condition must not be forgotten in our desire to get a good local result.

In the case of detachment occurring in an only remaining eye, the importance of regaining vision is sufficiently great to warrant the above indicated line of treatment, and there is seldom any difficulty in persuading the patient to submit to it. When the detachment is large and evidently much distended by the subjacent fluid, puncture of the sclerotic at the site of the greatest displacement may be resorted to as a preliminary to other treatment.

When there is reason to believe that the detachment is due to a choroidal tumour, excision of the globe without delay is the proper course.

Injuries of the Retina.—Blows on the eye may cause hæmorrhage in the retina, or detachment. Occasionally, when the eyeball receives a direct blow about the middle of the cornea, the macular region is injured, and becomes œdematous or slightly detached, with local choroido-retinal changes. It is not uncommon for the eye after a contusion to be almost blind for a short time, the sight then returning to normal without any ophthalmoscopic change being visible (retinal concussion). In rare instances of this kind we find a localised milky-white cloudiness of the retina corresponding to the part at which the force of the blow was felt. This lasts for about three days and then disappears, and soon afterwards the vision usually is restored to normal (*see also* "Eyeball, Injuries of," vol. iii. p. 207).

Glioma of the retina is essentially a disease of early childhood, and is usually met with at some time between infancy and the age of three years. It commences insidiously without pain or inflammation, and grows rapidly.

By its growth *forwards* it soon fills the eyeball, and then perforating the outer coats involves the other orbital tissues; finally, there is a very large, fleshy, readily bleeding protrusion from between the lids without any manifest trace of the globe. Backwards it extends along the optic nerve, and ultimately invades the brain. It not infrequently causes secondary deposits in the scalp, the neighbouring lymphatic glands, and sometimes in the liver and other distant parts. Death takes place from exhaustion or from implication of vital organs.

Occasionally it attacks both eyes independently. It sometimes occurs in several members of the same family.

Diagnosis.—Sometimes the child is brought at an early stage, because the attention of the mother or nurse has been aroused by seeing a whitish appearance from behind the pupil. At this time the eye is still quiet, looks natural, except for its white reflex, and has normal tension. In many cases the child is not brought until a later period, when redness and pain in the eye are observed; we now usually find the symptoms of secondary glaucoma—dilatation of the pupil, slight steaminess of cornea, engorged scleral veins, tenderness on light pressure, and increase of tension. By focal illumination we see a yellowish white, rounded, or lobulated solid-looking mass projecting into the vitreous, with blood-vessels visible on its surface, and occasionally small, white, chalky-looking bodies in the vitreous over it.

In some cases, especially in an early stage, it is simulated by inflammatory deposits occurring in the vitreous, due to suppurative choroiditis (pseudo-glioma). The tension of the eyeball in this affection, however, is never raised, and is usually found diminished. As all vision is lost, it is our duty, in every doubtful case, to deal with the eye as if it were a glioma.

Pathology.—The naked eye appearance is that of a soft, marrow-like growth of a whitish or reddish white colour, sometimes studded with small calcareous bodies; it is never pigmented. The tumour is composed of small cells, and the growth generally originates in the granular layers of the retina.

The *treatment* consists in early removal of the affected eyeball, dividing the optic nerve as far back as possible. If extra-ocular structures are already to some extent affected, it is advisable to thoroughly eviscerate the orbit, and apply chloride of zinc paste to the surface so left. If

the tumour is very large its removal cannot be expected to save or even prolong life, and it is advisable to leave it alone. In such a case palliative medical treatment is all that can be done.

Congenital Abnormalities.—The absence of pigment in albinos has already been mentioned. For the appearance of the retina met with in coloboma of the choroid, we must refer to the account given of this latter condition (*see* art. "Choroid," vol. ii. p. 139).

Not infrequently we find *small*, *round*, *black spots* on the fundus, usually few in number, and not interfering with normal vision. They have an appearance such as would be presented by tiny drops of black pigment flicked over the red fundus from a brush, and are due to localised aggregations of retinal epithelial pigment. In some cases, again, we may observe *very small bright dots* in the retina, chiefly in the neighbourhood of the optic disc. They can only be seen by the direct method, and are generally found near the large vessels in the optic disc neighbourhood, flashing into view and disappearing on small movements of the mirror, but always reappearing in the same position, and best seen near the edge of the image of the flame. Their presence is often accompanied by symptoms of asthenopia.

Opaque nerve fibres usually occur in the form of a wedge, having its blunt apex at the edge of the disc, and its base directed towards the equator of the eye. The affected area is brilliantly white, often rather fluffy in appearance, and the large retinal vessels are here almost, if not quite, concealed from view. It seldom reaches far peripherally, and generally ends in an irregular teased-out-looking border. It is commonly situated above or below the disc, but may completely surround it. Exceptionally, the patch of opaque nerve fibres is situated at some distance from the disc, and separated from the latter by normal red fundus; it is then more brush-like in form, the inner end presenting an abrupt edge, while its equatorial end is here again broader and teased-out-looking. The whiteness and opacity in all cases of this nature are due to the presence of a medullary sheath round the axis cylinders of the retinal nerve fibre layer. Normally, all these fibres gain this covering at the scleral opening, but exceptionally it is assumed earlier—in some few cases, as we have seen, to be lost again temporarily. Occasionally this retinal condition is confused with inflammatory effusion or choroidal atrophy; attention to the state of the fundus elsewhere, and to the pigmentary and vascular appearances, should leave no doubt as to the true nature of each of these several ophthalmoscopic changes.

Congenital irregularities in the blood-vessels of the retina are not infrequent. It is by no means uncommon to have the retina supplied in part by a branch from one of the posterior ciliary arteries, a so-called *cilio-retinal artery*. It generally pierces the disc near its edge, and then curves backwards to ramify over the adjacent retina, taking the place of a branch of the *arteria centralis*. In cases of embolism of the latter, the presence of a cilio-retinal vessel becomes important, as the portion of retina corresponding to this blood-supply retains its visual function.

Occasionally we find the retinal vessels very tortuous, simply as a congenital peculiarity. Very rarely a direct communication between a retinal artery and vein has been observed, and sometimes between a retinal and choroidal vessel.

Retinitis. *See* Retina and Optic Nerve (*Inflammation of Retina*).

Retino-Choroiditis. *See* Choroid, Diseases of (*Retino-Choroiditis*).

Retinoscopy.—Synonyms.—Shadow Test, Skiascopy, Keratoscopy, Pupiloscopy.

Retinoscopy is the most accurate and the most generally useful of the objective methods of estimating the refraction of the eye. By it can be determined the amount of hypermetropia, myopia, or astigmatism—both corneal and lenticular—that may be present, and while practising it, valuable information as to the condition of the media is obtained.

Of the two other objective methods, *direct estimation* by means of the refraction ophthalmoscope is one which should be practised whenever possible. By this method alone can differences in level in the fundus be accurately determined, while at the same time a detailed view of the fundus is obtained; but when measuring the refraction, even for the most expert, it is very difficult to get such an accurate result—especially in low degrees of astigmatism and the determination of the axes—as that obtained by retinoscopy.

The various *ophthalmometers* measure only the corneal astigmatism, and do not deal with either lenticular astigmatism or any spherical error of refraction (hypermetropia or myopia). Both these methods are valuable for estimating the refraction in certain cases.

The practice of retinoscopy is carried out as follows: The observer sits one metre in front of the patient in the dark room. The lamp is placed above and slightly behind the patient's head, so that its light falls directly on the observer and not on the patient's face. It is well that the room be as dark as possible, so that there may be no unnecessary reflections from the patient's cornea, and that the redo-reflex may be of maximum brightness.

By means of a perforated mirror held up to his eye, the observer reflects the light into the patient's eye, looking meanwhile through the

central aperture, when he sees at once the *red reflex*. The light from the mirror passes through the patient's pupil, and falling on the retina, illuminates a certain area. The light reflected from this area passes out from his eye to the eye of the observer, and the latter sees the patient's pupil brightly illuminated. By rotating the mirror on its axis the position of the light area is of course changed. If now the observer rotates the mirror, he will see a dark shadow—with a nearly straight edge at right angles to the direction of the movement of the mirror—pass across the patient's pupil; either in the same direction as—that is *with*—the mirror, or in the opposite direction to—that is *against*—the mirror. The edge of the shadow is the edge of the illuminated area, and the appearance of the shadow is due to the observer looking at an unilluminated portion of the retina.

It is upon the apparent movement of this light area with its attendant shadow in relation to the movement of the mirror that the method of retinoscopy depends.

The relative agreement or disagreement of the movement of the shadow and the mirror depends upon (*a*) the mirror that is used—plane or concave; (*β*) the refraction of the eye, and, in certain cases to be explained below, (*γ*) upon the distance of the observer from the patient.

Thus, the observer sitting at one metre from the patient with the *plane* mirror—

The shadow moves *with* the mirror in hypermetropia, emmetropia, myopia of less than 1D.

The shadow moves against the mirror in myopia of more than 1D.

With the *concave* mirror—

The shadow moves against the mirror in hypermetropia, emmetropia, myopia of less than 1D.

The shadow moves *with* the mirror in myopia of more than 1D.

The mirrors commonly employed are about 4 cm. in diameter. They should not have the glass perforated in the centre, since the edge of the perforation is apt to give a troublesome reflection, but merely an opening in the silvering about 3 or 4 mm. in diameter. The usual concave mirror has a focal length of 22 cm. Both the plane and concave mirrors have their special advantages, but perhaps the plane mirror is more generally useful.

To understand the relative movements of the shadow and mirror, we must trace the course which the rays of light take and find out.

I. How the movements of the mirror cause the light area to move *on the patient's retina.*

(*a*) When a plane mirror is used.

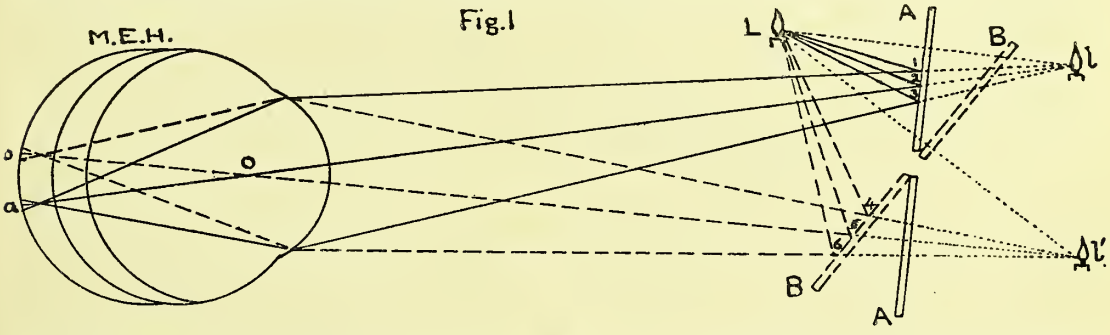
(*b*) When a concave mirror is used.

II. How this light area on the patient's retina appears to the observer to move—in (*a*) emmetropia; (*b*) hypermetropia; (*c*) myopia.

I. (*a*) Plane mirror.

In Fig. 1, AA is a plane mirror, L the source of light. Rays of light reflected from the mirror AA will appear to come from a point *l*, which may be called the *immediate source of light*, situated on the line L*l* drawn perpendicular to the mirror's surface, and the same distance behind the mirror that L is in front. The rays

Fig. 1



L1, L2, L3, being reflected into the eye MEH, will appear to start from *l*, and will be brought to a focus about the line *l*20, drawn from the immediate source of light through *o*, the optical centre of the eye, and strike the retina at *a*.

If now the mirror be rotated about its axis to the position BB, the immediate source of light will then be at *l'*. The rays L4, L5, L6, which enter the eye, will be brought to a focus about the line *l'*50, and will strike the retina at *b*.

Hence it is clear that as the mirror is rotated *upwards*, though the immediate source of light moves downwards, *the light on the patient's retina moves upwards*; also it will be seen that this will be the case whatever may be the refraction of the eye.

Thus the *light on the patient's retina always moves with the plane mirror in* E, M, *and* H.

(*b*) Concave mirror.

In Fig. 2, L is the source of light. AA is a concave mirror which brings the rays from L to a focus in front of the observed eye MEH. The rays L1, L2, L3, which are reflected into the eye, are made to converge, after reflection at the points 1, 2, and 3, to a focus at *l*. From *l* they will again diverge and pass into the eye. The focus *l*, then, is the *immediate source of*

light, and the rays will be brought to a focus about the line *lo*—the line joining the immediate source of light and the optical centre of the eye—and strike the retina at *a*.

If the mirror be rotated upwards to the position BB, the focus of the mirror must also move upwards, and thus the immediate source of light travels from *l* to *l'*. The rays L4, L5, L6, reflected from the mirror BB, will be brought to a focus at *l'*, and passing into the eye, will be focussed about the line *l'o* and strike the retina at *b*.

Thus, while the mirror is rotated upwards, the immediate source of light moves upwards,

Fig. 2.

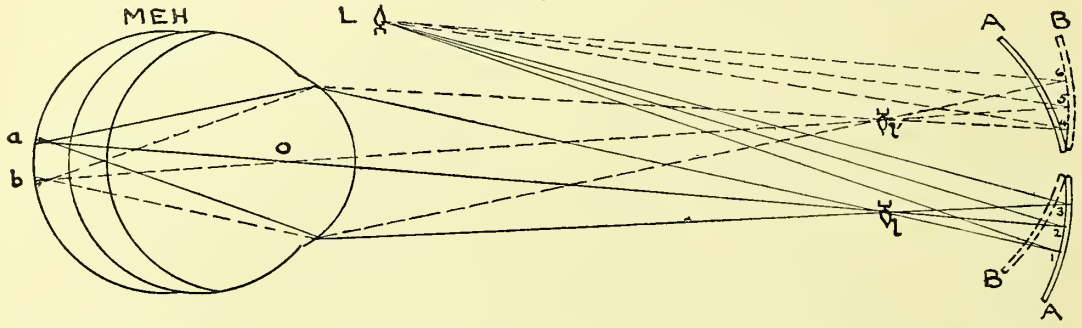
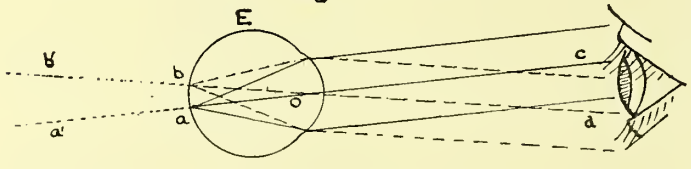


Fig. 3.



but the light on the retina moves *downwards*—that is, against the mirror, and this irrespective of the refraction.

The light on the retina, then, always moves against the mirror in E, H, *and* M.

If by holding the mirror very close to the patient's eye, or by using a mirror of long focal length, the rays of light do not cross in front of the patient's eye, then the light on his retina will move *with the mirror*, as if a plane mirror had been used. The writer constantly uses a concave mirror of 1·5 m. focal length, and sits 1 m. from the patient. The mirror then gives the same appearances as a plane mirror, with the double advantage that, the rays being made to converge, the light is brighter, and the troublesome shadow caused by the central aperture in the mirror is almost completely abolished.

II. We have now to consider how the light on the patient's retina appears to the observer to move.

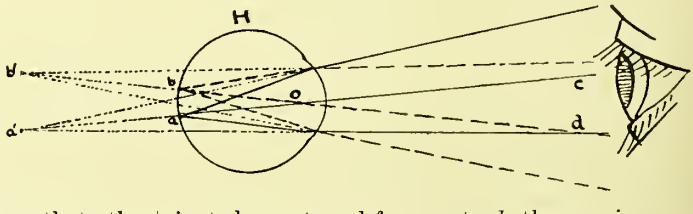
(*a*) *Emmetropia.*—In emmetropia parallel rays entering an eye are brought to a focus on the retina, therefore rays starting from the retina will emerge parallel.

In Fig. 3, E is an emmetropic eye—rays starting from an illuminated point *a* will emerge from the eye parallel to the line *aoc*; similarly, rays starting from *b* will emerge parallel to *bod*. The rays parallel to *aoc* which pass into the observer's eye will appear to him to come from a distant point, *a'*, behind the eye on the line *a'aoc*; similarly, rays parallel to *bod* will appear to come from a distant point, *b'*, on the line *b'bod*. As the light on the retina travels upwards from *a* to *b* the observer will see the light also move upwards from *a'* to *b'*. But we have seen that the light on the retina moves with the plane mirror and against the concave mirror. Hence *in* E the observer sees the light move *with the plane* and *against the concave mirror.*

(*b*) *Hypermetropia.*—In hypermetropia the retina lies in front of the focus for parallel rays, therefore rays starting from the retina will diverge as they pass from the eye.

In Fig. 4 the rays diverging from a point *a* on the retina of a hypermetropic eye, H, will appear to the observer to come from a point *a'* behind the eye on the line *a'aoc*. If the illuminated area travel from *a* to *b*, then again rays starting from *b* will appear to come from a point *b'* behind the eye on the line *b'bod*. Thus, as the light travels upwards the observer will see the light travel also upwards. Therefore *in* H, as in E, the observer sees the light move *with the plane mirror* and *against the concave mirror.*

Fig. 4.



(*c*) *Myopia.*—To understand how the observer

sees the light move in a myopic eye we must take—

(i.) A myopic eye *with more than* 1*D of myopia*—in which the far point[1] lies between the patient's eye and the observer—who we suppose is sitting one metre off.

(ii.) A myopic eye with less than 1D of myopia, in which the far point lies behind the observer.

(i.) Myopia of more than 1D.

Rays starting from the illuminated point *a*, on the patient's retina, are rendered convergent on leaving the eye, and meet at the far point *a'*. From *a'* they again diverge, and entering the observer's eye, will appear to him to come from this point. Similarly, rays starting from *b* will come to a focus at *b'*, from which they will appear to the observer to come. As the illuminated area on the retina moves from *a* to *b*, the source from which the observer sees the light come moves in the opposite direction, viz. from *a'* to *b'*; that is to say, the observer sees the light move in the opposite direction to which it is moving on the patient's retina. Thus, *in myopia of more than* 1D *the observer sees the light move against the plane mirror, but with the concave mirror.* (In this latter case it is seen that there is a double reversal of movement.)

(ii.) Myopia of less than 1D. If the far point be situated behind the observer's eye the rays will not have crossed before entering his eye,

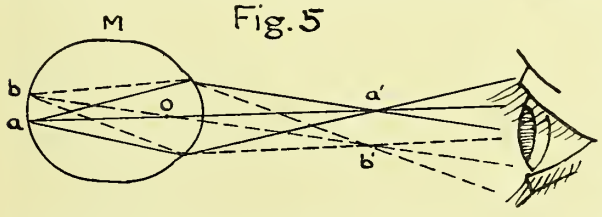


Fig. 5

and there will be no apparent reversal in the movement of the light. Therefore in this case the observer will see the light move in the same direction as it is moving on the patient's retina. Thus in *myopia of less than* 1D the light is seen to move *with the plane mirror* and *against the concave mirror*, as in H and E.

From the above considerations it is clear that between (i.) and (ii.) there must be a position of the far point at which, though the mirror is moved, the light and shadow do not appear to move at all; that is to say, the pupil appears either bright or dark, and no shadow is seen to move across it. What is this position? It is when the far point is situated at the observer's eye, or more accurately, when the rays from the illuminated retina are focussed exactly at his nodal point. This is termed the *point of reversal.*

This may be simply explained as follows:— Let A be the observed eye and B the observer's eye. The image of the pupil *ab* will be found on the observer's retina at *a'b'*.

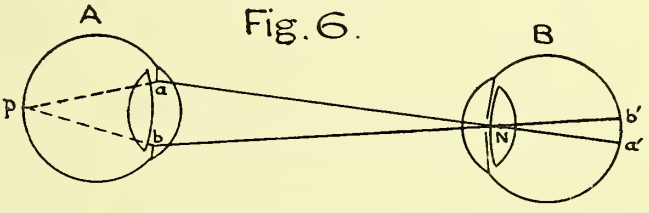


Fig. 6.

If the far point of the eye A lie at N, the nodal point of the eye B, rays from the very small area *p* (the size of which depends upon the size of the observer's pupil) will cover the whole area *a'b'*, and the observer therefore sees the whole of the patient's pupil illuminated. Thus, if *p* be illuminated the whole pupil will appear bright, if it be not illuminated the whole pupil will appear dark—hence at the point of reversal the whole pupil flashes either bright or dark—a condition of partial illumination of the area *a'b'* is impossible, and no shadow is seen to move across the pupil.

If then the observer's eye be 1 metre from the observed eye, and movement of the mirror causes no moving shadow, we know that the eye must have a myopia of 1D.

We are now in a position to understand the rule for estimating the refraction of an eye, viz. to place in front of the observed eye convex lenses in hypermetropia, and concave lenses in myopia, of gradually increasing strength, until the shadow ceases to move, when we know that the corrected eye has become myopic to the extent of 1D. We must deduct, therefore, 1D from the lens which abolishes the movement of the shadow to give the true correction of the error of refraction.

Thus, suppose it takes sph. + 4D to correct a hypermetropic shadow, that is to say, sph. + 4D gives the corrected eye 1D of myopia, the refraction of the eye is + 3D of hypermetropia; again, suppose it takes sph. − 5D to correct a myopic shadow, that is to say, sph. − 5D leaves the corrected eye with 1D of myopia, the refraction of the eye is − 6D of myopia.

It will be obvious from what has been said that the position of the observer is of great importance. We have seen that with the observer at 1 metre (the position of the far point of an eye of 1D of myopia), the glass

[1] The far point of an eye is the point for which the eye is focussed when accommodation is completely at rest, and therefore, conversely, rays starting from the retina are brought to a focus at the far point.

The F.P. of an eye having 1D of myopia lies 1 metre off.
,, ,, ,, 2D ,, ,, 0·5 ,, ,,
,, ,, ,, 0·5D ,, ,, 2 ,, ,,
that is to say, the distance of the far point varies inversely with the degree of the myopia.

which corrects the shadow leaves a myopia of 1D, but if the observer sits at 4 metres (the position of the far point of an eye of 0·25 of myopia), the lens which corrects the shadow will leave only 0·25D of myopia; similarly, when the observer sits at half a metre, the glass which corrects the shadow will leave 2D of myopia. The position of 1 metre is chosen for convenience, since glasses can easily be changed without the observer rising from his chair. The nearer the position chosen by the observer the greater is the error caused by unintentional variation from this position. Sometimes it is difficult to be certain which lens absolutely does away with the movement of the shadow; this may be tested by leaning forward a little, when the shadow should become "myopic," and again by moving back, when the shadow should become hypermetropic.

In order to appreciate the movement of the light area it is by no means necessary that the rays from this area be focussed on the observer's retina; indeed there is a considerable range in the position of the far point, both in front of and behind the observer's nodal point, in which it is quite impossible for a sharp image of the light area to be formed on the observer's retina, yet the movement of the light is always easily seen except at one point—the point of reversal.

Since the point of reversal depends upon the coincidence of the far point of the observed eye and the observer's nodal point, it is clear that, *provided the distance of his nodal point remain constant*, the same glass will be found to correct the movement of the light and shadow whether the observer be E, H, or M, and if ametropic, whether he wear his correction or not, or again whether he uses or relaxes his accommodation.

The brightness of the light area on the patient's retina, and the sharpness of the shadow, will depend chiefly upon the accuracy with which the light is focussed on the retina. When, therefore, the immediate source of light corresponds with the far point of the eye, the light area will be at its brightest, and the shadow most distinct.

The brightness of the light area and the sharpness of the shadow, *as it appears to the observer*, will depend upon the actual brightness of the light area, and also upon the degree of magnification. The magnification of the retina varies with the distance from which it is viewed. The farther the observer's eye from the point of reversal, the less the magnification; the nearer the point of reversal, the greater the magnification, until at the point of reversal the magnification is at the maximum.

This can easily be shown by looking at an object through a convex lens; when the lens is held close to the object the magnification is low; increase the distance and the magnification is raised, till at last one point only of the object is seen highly magnified. By removing the lens still farther a magnified reversed image is seen, which becomes smaller as the distance is still more increased.

On the degree of magnification depends also (*a*) *the rate of movement* of the light area and shadow (the rate of movement of the mirror remaining constant), and (*b*) the shape of the shadow.

With low magnification the shadow appears to move *slowly*, and a larger portion of the edge of the light area being seen, the shadow appears *curved*.

With high magnification the shadow seems to move *faster*, and as only a small portion of the edge of the light area is seen, much increased in size, the shadow appears *straight*.

Hence in high degrees of uncorrected H and M, where the magnification is low the light area appears dull, the shadow is curved and moves slowly.

As the point of reversal is approached the magnification increases, and the light area appears brighter, the shadow moves faster and has a straight edge.

Regular Astigmatism.—In regular astigmatism the refractive power of two meridians at right angles to one another is different. There must be, therefore, two far points, one for each meridian. In measuring the astigmatism by retinoscopy, the refraction of each meridian must be estimated separately, and the difference between the two correcting lenses will be the measure of the astigmatism.

Thus, suppose the vertical meridian be corrected by a lens of sph. +4D, and the horizontal meridian by a lens of sph. +6D, with the observer sitting at 1 metre from the patient; then sph. +4D combined with cyl. +2D axis vertical will give the patient 1D of myopia; the true correction, therefore, of his refraction will be sph. +3D cyl. +2D axis vertical.

Again, suppose the vertical meridian be corrected by sph. +1·5D, and the horizontal by sph. −0·5D, then either sph. +0·5D cyl. −2D axis vertical or sph. −1·5D cyl. +2D axis horizontal will give the true correction. *N.B.*—The cylinder must be placed with its axis at right angles to the meridian which it is to correct; it does not affect the meridian corresponding with its axis.

When the point of reversal for one of the meridians is nearly reached, it will be noticed that the light area has a *band-like* appearance, and that the direction of the band remains constant whatever the movement of the mirror may be. The axis of the band indicates the axis of that meridian; which observation is of great practical use when ordering glasses for children, with whom the suggestive test is unreliable. The reason for this band-like appearance is that the light area is magnified to the maximum by the meridian at whose point of reversal the observer's eye is, and therefore

to a much greater degree than by the opposite meridian. The light area is thus drawn out, as it were, and appears as a narrow band of light with a straight shadow on either side.

Double Shadows.—It is commonly found that a given lens will correct the shadow in the periphery, while there still remains an uncorrected shadow in the centre of the pupil, or *vice versa.* That is to say, the centre of the pupil appears as it were more hypermetropic or more myopic than the periphery. Of the two conditions the former is more common, and is due to the spherical aberration of the refractive media. In common with all spherical lenses, the periphery is more refractive than the central part, hence the centre may appear hypermetropic, while a myopic shadow is seen in the periphery. In these cases *always correct for the movement of the brightest part of the central light area,*[1] since it is through the centre of the pupil that the patient looks.

In conical cornea, lenticonous, and other allied conditions, the central shadow appears myopic, while the periphery appears hypermetropic; here again, as a rule, the movement of the central light area is the one to be corrected.

In *irregular astigmatism* a variety of conflicting shadows is often found; and though in some cases retinoscopy may lead to a right estimation, in others the subjective method of trial with lenses must be resorted to.

Mydriatics.—In order to obtain the most accurate estimation of the refraction by retinoscopy, the accommodation of the patient must be completely at rest, and he should look at the hole in the mirror in order that the light may play over the macular region. This is the part of the retina for which the correction is needed, and the refraction of the macula may be different from that at other parts of the fundus. Atropia undoubtedly gives the most complete relaxation of the accommodation, but the effects last for about ten days, and in elderly people the danger of inducing glaucoma must never be forgotten.

For children with active accommodation, and to whom time is as a rule of no object, a 1 per cent solution of atropin may be used three times a day for three days previous to examination. If atropin be contraindicated, homatropin and cocain may be used in—(i.) Discs containing gr. $\frac{1}{50}$ of each; or (ii.) A 2 per cent solution of each in castor oil (*N.B.*—One application only should be made, and only a very small quantity should be applied, and this should be spread over the cornea by means of the upper lid; an ordinary-sized drop causes a disturbing film over the cornea); (iii.) Many prefer a 2 per cent watery solution instilled four times at intervals of ten minutes. (Occasionally the cocain causes a roughening of the corneal epithelium. To avoid this it has been recommended to use the H. and C. solution the first and fourth time, and a 2 per cent solution of homatropin alone for the second and third installation.)

In whichever form the H. and C. be applied, the full effect is gained in about forty minutes. The effect passes off in about twenty-four hours, but if after examination a drop of a 1 per cent solution of eserin be instilled the mydriasis passes off in less than an hour. In elderly people with very small pupils, whose accommodative power is slight, sufficient dilatation of the pupil may be gained safely with a 2 per cent solution of cocain, or one application of H. and C., followed by a drop of 1 per cent solution of eserin.

It is well before ordering glasses to test the patient again with the trial lenses, after the effect of the mydriatic has passed off, *except in the case of children.*

Some deduction has, as a rule, to be made from the spherical lens (not the cylinder), which gives the full correction under the mydriatic. In children 1D may be deducted from the full atropin correction, except in the case of convergent strabismus, when only 0·5D should be deducted. Adults vary considerably; some will accept their full correction, others require even 1D to be deducted. Where it is impossible to test a second time, it is usually safe to deduct 0·75D or 1D atropin, as in children, and 0·5 for homatropin and cocain. (For special correction of myopia, see article on "Myopia.")

Retinoscopy without a Mydriatic.—In adults mydriatics may be undesirable for one reason or another, and in many cases retinoscopy without their use, combined with a careful subjective test, will give excellent results. Certain points, however, need specially to be considered. If a patient, not under the influence of a mydriatic, look straight at the sight-hole of the mirror two difficulties at once arise: firstly, he will use his accommodation; and, secondly, his pupil will become so small that the movement of the shadow can hardly be distinguished. The patient should therefore relax his accommodation by looking to the other end of the dark room past the observer's *right* ear, when his *right* eye is being examined, and past the observer's *left* ear when the *left* eye is under examination; the observer meanwhile must use his right eye when examining the patient's right eye, and his left for the patient's left eye. In this position the light is thrown by the mirror on the optic disc—the least sensitive part of the fundus—and the pupil therefore does not con-

[1] Throughout this article the movement of the light area with its attendant has been described for the sake of clearness as the object for observation. The writer would strongly urge the advisability of watching the movement of the brightest part of the illuminated area, seen about the centre of the pupil; as the shadow is at times very indefinite, while the movement of the light area may be quite distinct, and this is especially seen in the above-mentioned cases with double shadows.

tract so much, and at the same time the reflex is brighter. The error introduced is that the refraction at the disc may not be the same as at the macula, but with careful subjective testing this error may as a rule be eliminated. If, however, any difficulty be experienced a mydriatic should be used.

Retraction.—The act of drawing back or aside, *e.g.* the separation of the lips of a wound or of the vulvar aperture by a special instrument (a *retractor*); or, more specially, the contraction of the uterus which follows the birth of the child, and which is, unlike contractions in labour, not followed by complete relaxation. *See* Puerperium, Physiology (*Involution of the Uterus*).

Retraction-Ring. *See* Labour, Physiology of (*Factors of Labour*, *Primary Powers*, *Uterus*).

Retrahens.—Drawing back, as in the terminology of the muscular system; *e.g.* the *retrahens aurem* muscle (auricularis posterior), one of the extrinsic muscles of the ear, which, if well developed, draws the pinna backwards.

Retro-.—In compound words *retro-* means backward, as in *retroauricular* (behind the ear), *retrobuccal* (belonging to the posterior part of the mouth), *retrocedent* (disappearing from superficial and reappearing in internal parts, *e.g.* some forms of gout), *retrocession* (the act of going back, *e.g.* the fœtal head at the vulva in labour), *retroplacental* (behind the placenta), and *retrosternal* (behind the sternum).

Retrobulbar Neuritis. — Inflammation of the orbital part of the optic nerve; retro-ocular neuritis. *See* Amblyopia (*Toxic*, *Diagnosis*); Retina and Optic Nerve (*Inflammation of Optic Nerve*).

Retroflexion. *See* Uterus, Displacements of the (*Retrodisplacements*); Pregnancy, Affections of Generative Organs (*Retroversion of Gravid Uterus*).

Retro-ocular Neuritis. *See* Retrobulbar Neuritis.

Retroperitoneal. *See* Abdominal Abscess (*Retroperitoneal*); Abdominal Tumours, Diagnosis of (*Retroperitoneal Glands*); etc.

Retropharyngeal. *See* Pharynx, Diseases of (*Retropharyngeal Abscess*).

Retropubic. *See* Pregnancy, Affections of Generative Organs (*Retroversion of Gravid Uterus*, *Diagnosis from Retropubic Abscess*).

Retropulsion.—A driving backward, *e.g.* retropulsion of the coccyx during the birth of the fœtal head, or the tendency to run backward seen in the gait of paralysis agitans.

Retrotarsal.—Behind the tarsus of the eye, *e.g.* the *retrotarsal fold.*

Retrouterine.—Behind the uterus, *e.g.* a retrouterine tumour.

Retroversion. *See* Uterus, Displacements of (*Retrodisplacements*); Pregnancy, Affections of Generative Organs (*Retroversion of the Gravid Uterus*).

Retzius, Space of.—The cavum Retzii, a hollow on the pelvic aspect of the visceral pelvic fascia bounded laterally by the puboprostatic ligaments; in this space the bladder is not covered with peritoneum.

Revaccination. *See* Smallpox (*Treatment*, *Prevention*); Vaccination.

Reverdin's Method.—A method of epithelium grafting; also, a method of treating symblepharon. *See* Skin Grafting (*Methods*).

Reversion. *See* Heredity (*Reversion*).

Revulsives.—An almost obsolete term for agents believed to divert a disease from one part of the body to another, especially by blood-letting.

Rhabditis.—A filaria; *e.g.* *rhabditis Niellyi* (the parasite of craw-craw), *rhabditis intestinalis s. stercoralis* (*anguillula stercoralis*).

Rhabdo-.—In compound words *rhabdo-* (Gr. ῥάβδος, a rod or wand) means rod-like; *e.g.* *rhabdonema strongyloides* (*see* Parasites, *Nematodes*, *Angiostomidæ*), *rhabdomyoma* (a form of myoma containing striped muscular fibres).

Rhachi-.—In compound words *rhachi-* (Gr. ῥάχις, the backbone) means relating to the spine; it is often spelt *rachi-* (*q.v.*).

Rhachitis. *See* Rickets.

Rhagades.—The plural of *rhagas*, a fissure or tear (Gr. ῥαγάς, a rent or chink).

Rhamni Purshiani Cortex. *See* Cascara Sagrada.

Rhatany. *See* Krameriæ Radix.

Rheinfelden. *See* Balneology (*Switzerland*, *Muriated Waters*).

Rheonome, Rheostat, etc. *See* Electricity; Physiology, Tissues (*Muscle in Action*).

Rheumarthritis.—Rheumatic inflammation of a joint. *See* Rheumatism, Acute and Chronic.

Rheumatic Fever. *See* Rheumatism, Acute.

Rheumatism.—This subject is discussed under the following sections:—

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4. Chronic Rheumatism . . . 573

See also Adrenal Glands, Addison's Disease (*Diagnosis, Pigmentation in Rheumatoid Arthritis*); Alcohol (*Indications in Rheumatism*); Anæmia (*Causal Factors, Rheumatism*); Auditory Nerve and Labyrinth (*Nerve-Deafness, Toxic*); Balneology (*In Disorders of Nutrition*); Bone, Diseases of (*Osteomyelitis, Diagnosis*); Brain, Affections of Blood-Vessels (*Cerebral Embolism, Causes*); Bronchi, Bronchitis (*Acute, Etiology*); Bursæ, Injuries and Diseases of (*Bursitis, Rheumatic*); Children, Clinical Examination of (*Neck, Stiffness*); Chorea (*Etiology*); Cornea (*Localised Keratitis, Causes*); Deformities (*Flat-Foot, Diagnosis; Metatarsalgia, Diagnosis*); Dengue (*Diagnosis*); Diathesis (*Rheumatic*); Dysentery (*Diagnosis*); Eczema (*Etiology*); Electricity (*Electric Bath in Rheumatism*); Hæmoglobinuria (*Causes*); Hæmophilia (*Diagnosis, Joint Affections of Rheumatism*); Heart, Myocardium and Endocardium (*General Pathology, Etiology*); Hip-Joint, Diseases of (*Acute and Chronic Rheumatism*); Hydropathy (*Acute Rheumatism*, etc.); Hypnotism (*In Rheumatism*); Hysteria in Childhood (*Etiology*); Insanity, Etiology of; Insanity, Nature and Symptoms (*Etiological Classification*); Iris and Ciliary Bodies (*Inflammation of Iris, Rheumatic Iritis*); Joints, Diseases of (*Constitutional Conditions*); Larynx, Neuroses of (*Laryngeal Paralysis, Varieties*); Menopause (*Associated Disorders, Rheumatoid Arthritis*); Menstruation and its Disorders (*Dysmenorrhœa, Varieties, Systemic Causes*); Meteorology (*Influence on Health, Cold and Damp*); Miliaria (*Acute, Causes*); Nails, Affections of the (*In Acute Rheumatism*); Nerves, Peripheral (*Neuritis, Causes*); Nerves, Multiple Peripheral Neuritis (*General Etiology*); Nerves, Neuralgia (*Etiology*); Ocular Muscles, Affections of (*Paralysis, Etiology*); Pericardium, Diseases of (*Pericarditis, Etiology*); Peritoneum, Acute Peritonitis (*Clinical Varieties, Rheumatic*); Pleura, Diseases of (*Pleurisy, Etiology*); Pulse (*Rate and Temperature in Rheumatic Fever*); Sclerotic, Diseases of (*Sclerotitis, Causes*); Scrotum and Testicle, Diseases of (*Orchitis, Causes; Testalgia, Causes*); Septicæmia (*Differential Diagnosis*); Shoulder, Diseases and Injuries of (*Rheumatoid Arthritis*); Spine, Surgical Affections of (*Spondylitis Deformans*); Syphilis (*Secondary, "Rheumatic Pains"*); Tabes Dorsalis (*Sensory Symptoms, Pains*); Teeth (*Dental Caries, Causes, Saliva in Rheumatism*); Teeth (*Diseases of Peridental Membrane, Etiology*); Tetany (*Pathogeny*); Therapeutics, Health Resorts (*Climate for Rheumatism*); Thyroid Gland, Medical (*Acute Thyroiditis, Causes*); Trades, Dangerous (*Mercurialism, Diagnosis*); Undulant Fever (*Diagnosis*); Urticaria (*Diagnosis*).

Acute Rheumatism

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Rheumatic Fever is a disease extremely difficult to define, and yet, when it occurs in its usual types, has all the characters of a specific affection. It is when the unusual forms of the disease are studied that we are led to recognise that it is one of a large group of diseases, each member of which possesses definite characters, but all of which bear a certain family resemblance to one another. The malady is a very serious one, and in the crowded cities of England very common; but because it is not often directly fatal, the serious results that arise are liable to be imperfectly realised. Again and again we see, especially among the hard-working classes, the young, the attractive and intelligent, maimed by serious heart disease, and can trace from a first attack of rheumatic fever the commencement of a life of suffering brightened only by brief intervals of respite.

Etiology.—The causes may be divided into the predisposing and exciting.

Predisposing Causes.—Inheritance is of much importance, and double inheritance is more liable to result in a severe attack in the child than is heredity on one side only.

It is probable that there is in rare instances direct infection of the fœtus through the placental circulation, and there is little doubt that some cases of congenital heart disease are due to this direct infection, though more usually there would appear to be not a direct infection, but an inherited vulnerability of the tissues.

Rheumatic fever is more common in childhood and adolescence than in adult life; and not only this, the disease is more virulent and the manifestations more numerous in the young. One attack does not confer immunity, and there appears to be in many instances an increased liability to further attacks. On the other hand, with advance in age the resisting power of the constitution increases, and in considering immunity we must remember that it is extremely difficult to feel sure when an attack of rheumatic fever is absolutely cured.

The influence of sex is inconsiderable. The season of the year has a marked effect: in autumn and spring rheumatic fever is rife, and if the autumn commences with cold and wet,

after a long spell of dry and hot weather, numerous cases are sure to be met with in the towns. As Dr. Newshohme has insisted, rheumatic fever is an urban disease, and he associates it also with a low level of ground water.

Mental and physical strain are additional factors. There is good reason to believe that the strain of education, examinations, and the contest for medals among the underfed and more intelligent of the children of the poor, assist in the causation of the choreic type of rheumatic fever.

Sudden chill, especially if with this there is physical overstrain, is a powerful predisposing cause, and it is not uncommon to get a very definite history of such an occurrence from which the attack can be accurately dated. Acute rheumatism appears to be ubiquitous, but in warm and equable climates its occurrence is not so frequent, and it is not unlikely that the thorough realisation of rheumatic fever as an infective disease will bring to light some important facts upon this question of climatic influence.

Epidemics have been recorded by Dr. Mantle and others, but they are unusual.

That any particular diet predisposes does not seem probable, and rests on no very sure evidence. Insufficient food, the emanations of sewer gas, and overcrowding, by lowering the health, are predisposing causes, and of these foul air is probably an important one.

The Exciting Cause.—Rheumatic fever is an infective disease, and as in other diseases of this type there are many factors which predispose to the actual infection, so, too, the same is true in rheumatic fever. The question also arises whether, in addition to various predisposing factors, there are several exciting causes. The answer to this question cannot yet be said to be decided, though, as the result of my researches with Dr. Alexander Paine, I believe that one and not several different bacteria will prove to be the true excitant of rheumatic fever. This in any case may be asserted with confidence, that the excitant is a bacterial infection; and that the view of the infective origin of the disease has not been the result of a sudden desire to keep, as it were, in the fashion, but has been the outcome of many years of investigation by clinicians, pathologists, and bacteriologists, and that by this view the manifestations are well explained.

At first the results of bacteriological investigation were indecisive, but of late Triboulet and Coyon, Wassermann, Poynton and Paine, and Fitz-Meyer have independently isolated diplococci which, though differing in some minor respects, appear to be identical and to be a cause of the disease. The earlier researches of Wilson, Mantle, von Leyden, Löffler, and many others prepared the way for these later investigations. The micrococcus, as described by Dr. Paine and myself, is minute, and essentially a diplococcus, each element of which is ·5 μ in diameter. On solid media it grows in staphylococcal masses, in liquid media in streptococcal chains, each element of which is a diplococcus. We found the most favourable culture medium was blood-agar, or milk and bouillon slightly acidified with lactic acid. We have isolated it from twenty cases of rheumatic fever. It is present in the local lesions, and in the blood and urine of the living patient suffering from pericarditis. Injected intravenously into rabbits it reproduces the disease, and can be isolated from the local lesions. The organism is rapidly destroyed at the sites of the local lesions in man, and this fact, together with its minute size, makes it difficult to find in the tissues. We have succeeded in demonstrating the diplococcus in the nodules and in the synovial membranes of the joints, and have isolated it from both these structures and from most of the rheumatic lesions. It is usually present in the exudations, though in very acute and clear effusions it may be absent, or present only in small numbers.

In suitable media it lives for long periods, and keeps in some measure its virulence. It stains well with the aniline dyes, but in the tissues does not retain the gentian violet used in Gram's method with tenacity.

The diplococcus certainly gains access to the system through the tonsils, and may also do so by other paths which have not as yet been demonstrated.

On the other hand, Singer maintains that rheumatic fever is an attenuated pyæmia due to infection with various pus-forming cocci. But it appears inadvisable to consider such a constant disease as rheumatic fever as an attenuated pyæmia until several specifically different pus-forming micrococci are proved conclusively to cause rheumatic fever.

A brief allusion must also be made to a view which maintains that arthritic rheumatism is the true disease due to an unknown excitant, and that pericarditis and the other severe lesions are the result of secondary infections. To consider rheumatic fever as a multiple arthritis, or the severe lesions as complications, is, in our opinion, contrary to clinical experience, for they are manifestations of the disease in its severe forms; further, the diplococcus may be present in the synovial membranes of the joints although the clear exudation may be sterile, and hence these lesions, which are looked upon by upholders of the view as true rheumatism, are identical in their origin with other rheumatic manifestations.

Although there are such strong reasons for believing the cause of rheumatic fever to be an infection with a diplococcus, this can only explain one factor in the disease—the exciting cause. The peculiar poisons that are formed,

and the peculiar vital chemistry of the human tissues, are as yet unknown.

The Morbid Anatomy.—In studying the morbid anatomy of rheumatic fever we are struck by the numerous local lesions that are found, and the disease in this respect is in marked contrast to diphtheria, in which the local lesions are but few. In these positions the diplococci are rapidly destroyed by the cells of the living tissues, but while present in these lesions they give rise to a condition of inflammation which often leaves the tissues more or less damaged. These various inflammations in rheumatic fever rarely suppurate, but when the tissue changes are severe the process of recovery is incomplete, and sclerosis and consequent deformity of such delicate structures as the cardiac valves will result.

The recovery from arthritis is usually complete, and nodules may entirely disappear, but inflammation of the valves and pericardium repeatedly ends in deformity of the valve segments and adhesion of the pericardium. So also in some cases the joints do not completely recover, and the nodules may persist for many months. We are prepared—since the local changes are the result of the invasion of the diplococcus—to find that the various lesions of rheumatism are all of the same type, when due allowance has been made for the anatomical peculiarities of the various tissues.

If, for example, rheumatic valvulitis is chosen to illustrate those changes, they will be found to be the following:—*Microscopic.*—When the diplococcus gains access to the valve, the connective tissue immediately beneath the endocardium swells, and the fibres become indistinct and separated by a slight exudation. Thus the endocardium at that point is raised above the general surface of the valve. Later the connective tissue cells proliferate, the endothelial lining is destroyed, the destructive process advances to the stage of necrosis, and numerous leucocytes will be found in the damaged tissues. Thus the vegetation is formed, and usually there are a series of these fringing the edge of the segments of the valve. To these vegetations fibrin often becomes attached, but this deposit of fibrin is no essential factor in their formation. Repair commences by invasion of the necrotic part of the granulation by fibroblasts, and thus eventually sclerosis and deformity of the valve result. It is an important fact that by the time the vegetation is completely formed most of the organisms are destroyed by the activity of the tissue cells and leucocytes.

The macroscopic appearances of rheumatic valvulitis vary with the stages of the process. In the acute stage, if the valve was previously uninjured, minute reddish pink elevations are visible, and the valve itself is swollen. When the process is less acute and the valve damaged by earlier attacks, minute yellow, somewhat translucent excrescences are formed. After repeated attacks of inflammation the sclerosis of the segments of the valve which results is only too well recognised. The mitral valve is the most frequently, and also the most extensively injured. Next in order comes the aortic valve, then the tricuspid, which in childhood is affected more often than is generally thought, and, finally, though rarely, even the pulmonary valve may show slight beading.

The nodule passes through a like series of changes; under the microscope in an early nodule the zones of necrosis, cell proliferation, and swollen connective tissue can be readily traced, and in the interstices of the swollen connective tissue the diplococci may be demonstrated. In their macroscopic appearance these nodules resemble a minute sago grain, but the more carefully an attempt is made to dissect them from the tissues, the more surely do they disappear under the knife, for the exudation within them is pressed out, and scarcely a trace of the nodule can then be seen. They are not therefore in their characteristic stage *fibrous* nodules.

When the diplococci attack the more vascular structures, such as the pericardium or joint capsules, they locate themselves in the delicate areolar tissue immediately beneath the endothelial lining. Great dilatation and often rupture of the capillary blood-vessels result, and there is the same cell proliferation, leucocytic invasion, and connective tissue swelling as already described. The exudations into the pericardial sac or joint cavities are at first clear or blood-stained, and when the endothelium is intact are usually sterile. This latter fact is also confirmed by experiment, which is important, for investigators have experienced difficulty in isolating the organism from these clear effusions.

Later the fluid becomes more opaque and fibrino-cellular, and in some cases layers of the plastic exudation are deposited upon the inner surface of the pericardium and synovial membrane. A film of these later exudations will show under the microscope numerous leucocytes, which for the most part stain well with aniline dyes and are not necrotic. The interstitial tissue between the muscle bundles in the neighbourhood of the inflamed joints is also the seat of similar changes. The tendon sheaths may suffer in exactly the same way, and the morbid changes in the pleura are also of the same character.

The macroscopic appearances of the pericardium, pleura, and joint capsules are much alike, although, since it is the virulent rheumatism in which the pericardial tissues are especially attacked, the changes in that structure are usually more definite. In the early stage there is swelling, the tissues on section look gelatinous, and where the blood-vessels are

chiefly distributed there is much hyperæmia. Later there may be sclerosis and adhesion.

The rheumatic poisons are injurious to the myocardium. In severe cases there is considerable fatty degeneration of the fibres, and though this change is most evident beneath the inflamed pericardium, it occurs throughout the heart wall, and is especially noticeable in the neighbourhood of the blood capillaries. In some cases there may also be considerable cell exudation in the fibrous tissue between the muscle bundles, and when the process is long continued a definite fibrosis of the heart wall results.

Acute arteritis has been described by Legroux as a result of rheumatic fever. The changes commence immediately beneath the lining of the vessel wall, and are of the usual type.

Another morbid change in connection with the cardio-vascular system is a fibrosis around the arterioles and capillaries, and is of interest as seeming to throw some light upon the pathology of arterio-capillary fibrosis.

Phlebitis with consequent thrombosis may also result from the rheumatic process. There is absence of suppuration, and in severe cases after the process has subsided the clot is organised and the vessel converted into a fibrous cord.

The study of the morbid anatomy of such viscera as the lungs, the liver, the spleen, and the kidneys is a more difficult matter. There are the complicating factors of cardiac failure from mechanical embarrassment due to injury of the valves and consequent "backworking." There is also the fortunate rarity of death from a first attack. We are cramped, too, by such familiar terms as "nutmegged liver," "mechanical congestion of the lungs," and "cyanotic induration of the kidneys." Yet there are good reasons to believe that, apart from the well-recognised and definite changes due to cardiac failure, there is also another series of changes in these viscera, the direct result of the rheumatic infection.

The Lungs.—Pneumonia, for example, is one of this series.

Rheumatic pneumonia is broncho-pneumonic in its distribution and seldom massive, as is the pneumonia due to the diplococcus lanceolatus. The patches are generally near the pleural surface of the lung, though in some cases they are scattered throughout. There is some amount of pulmonary collapse around these areas of pneumonic change. The blood-vessels are greatly distended, and there are often hæmorrhages in the alveoli, which also contain endothelial cells and leucocytes, but, as a rule, not a well-marked network of fibrin.

The diplococci, which are very small and not lanceolate, are sometimes present in great numbers in the exudation.

Apart from mechanical congestion of the lungs, a definite pulmonary œdema sometimes occurs which is most marked in the upper lobes. The lung is firm, but doughy on pressure, and on section much œdema fluid can be squeezed from the cut surface. Microscopic examination shows that the alveoli are full of this œdema fluid.

The Liver.—In a fatal first attack of rheumatic fever, when there is no œdema or other definite evidence of "backworking," the liver will be found uniformly pale, and a section of such a liver shows extensive fatty changes, which have not the distribution characteristic of the "nutmeg liver." The change is general, and most probably the direct effect of the rheumatic poison. The occurrence of hæmatoporphyrinuria has, from quite another line of evidence, been pointed out by Dr. A. E. Garrod as extremely suggestive of alteration in the hepatic metabolism in rheumatic fever.

The Spleen.—In some of the more malignant types the spleen, as pointed out by Professor Litten, is uniformly enlarged, without a trace of infarction to account for it. But, as a rule, the spleen does not seem to be one of the viscera which is much altered by the direct effect of rheumatic fever.

The Kidneys.—In severe cases there may be well-marked cloudy swelling, coagulation necrosis, and fatty change in the cells of the convoluted tubules of the kidneys, and it is very probable that the toxins are excreted by this channel and are the cause of these changes. Hæmaturia, apart from infarction, may result in the malignant types of the disease, and albuminuria may also occur, both of which occurrences lend support to the view that the rheumatic poisons have a direct action upon the kidneys, though in man this is not usually of a severe type. The diplococcus can be isolated from the urine in virulent cases of rheumatic fever.

The Brain.—The morbid changes in rheumatic chorea are extremely difficult to ascertain, the opportunities are so rare, and the brain so difficult to thoroughly investigate. Hyperæmia and minute hæmorrhages, exudation around the capillary blood-vessels, cloudy swelling of the neurones, sometimes minute emboli or thromboses, and a slight leptomeningitis, are among the changes observed, and are such as we should be led to expect.

After death from hyperpyrexia, as a rule, no definite morbid changes have been found in the brain.

Angina Faucium.—The faucial inflammation is in no way distinctive; it is usually catarrhal and rarely suppurative, but there may be a membranous deposit upon the tonsils or a condition of follicular tonsillitis. From repeated attacks considerable fibrosis of the tonsils results, and a section through such tonsils may show loculi containing inflammatory exudation at some distance from the surface.

The diplococcus gains access to the system

through the tonsils, and may be found in the interstitial framework in the neighbourhood of the small blood-vessels; but to isolate them from the numerous micro-organisms that are always present in the throat is a very difficult undertaking in the present state of our knowledge.

The Cutaneous Lesions.—Apart from the sudamina and miliaria, there is in most of the rheumatic lesions great dilatation of the blood capillaries, and often, as in purpura, actual rupture of their walls. In addition, in some cases there is considerable exudation into the subcutaneous tissues, a change which is especially noticeable in erythema nodosum.

Infarction.—The occurrence of infarcts in the viscera in rheumatic fever is an interesting problem. They occur far more frequently in malignant endocarditis, and are ascribed to the detachment of portions of the exuberant vegetations or to detached blood-clot. In simple rheumatism they occur most usually in advanced mitral lesions.

The Blood.—There is usually well-marked coagulation of the blood after death.

Symptoms and Course.—Rheumatic fever has no definite incubation period, and the onset may be either acute or, as is more usually the case, gradual. When the onset is acute there is some definite history of chill or exposure from which the illness is dated, and the first symptoms generally show themselves within a week.

There is a feeling of malaise, with moderate fever and chilliness; there may be a sore throat or stiff neck, and the eyes look dull and heavy. Muscular feebleness, fleeting pains in the limbs, and prostration follow, and usually in the adult acute multiple arthritis develops and makes the diagnosis clear. When the onset is gradual there are often vague symptoms, which, now the disease is recognised as infective, are of much interest. Anæmia, irritability, loss of energy, and wasting are frequent, especially in young subjects. Again, headache, epigastric pain, and stiffness in the muscles and joints are very suspicious symptoms, the more so if they are accompanied with a slight rise of temperature.

The acute attacks of rheumatic fever in adult life we associate with a rapid and transient multiple arthritis, which quickly renders the patient quite helpless, and at the same time makes the nature of the illness apparent. Yet it is unfortunately the case, that with them as with children, the first unequivocal sign of rheumatic fever may be inflammation of the heart and not arthritis, and this carditis may have been in existence for some days before precordial pain and breathlessness convince the patient that he is seriously ill.

A severe attack of the arthritic type of rheumatic fever is at its height a very characteristic disease. The sufferer lies in bed quite helpless, dreading the slightest movement; many of the larger joints, and not infrequently the smaller ones as well, are swollen and extremely tender. Over the joints, which feel hot to the touch, there may be a red flush, and the implication of the tendons and the periarticular tissues in this transitory inflammation add to the distress.

From day to day joints which were previously unaffected are attacked, and others already inflamed begin to subside often with great rapidity.

The temperature is raised to a moderate degree (102°), but runs no definite course. The mind is clear and as a rule acute; the tongue is thickly coated with a white fur. In many cases there is profuse sweating, and the sour odour that is present has long been pointed out as a feature of the illness. If the sweating is profuse there is considerable thirst, but the appetite is lost, and bowels are often constipated. The pulse varies with the extent of the implication of the heart, but it is usually increased in rate, regular, with a large wave, and easily compressible. Examination of the heart very frequently shows that there is a definite dilatation. The impulse is diffuse; the area of deep cardiac dulness increased, especially toward the left; the first sound of the heart over the region of the impulse is short, and the second sound over the pulmonary area accentuated. In addition there may be a soft systolic murmur heard most plainly internal to and slightly above the left nipple.

This represents the earliest affection of the heart in rheumatic fever, and this eminently recoverable condition may drift almost imperceptibly into organic valvular disease. The clinical importance, therefore, of this dilatation is, as Dr. D. B. Lees has insisted, great.

The *respiratory system* is but little affected. Except for anxiety and distress of mind, with insomnia from the pain, there are no definite *nervous* disturbances. *The urine* is scanty, high-coloured, and strongly acid; the chlorides are diminished, and the urates are increased and form a thick deposit. Hæmatoporphyrin is sometimes present in large quantities.

The course of the illness is very indefinite, and may be protracted by repeated relapses over many weeks. An acute attack lasts, as a rule, about three weeks, but at the present time active treatment is so commonly adopted that it hardly serves a useful purpose to lay down any definite duration for the illness. Rheumatic fever is one of those diseases from which convalescence is often slow, and in which it is extremely difficult to feel sure when all active symptoms are at an end. Recovery finds the patient anæmic and prostrate, and it is not uncommon to find that the joints remain stiff and muscles tender for a long time after the recovery is otherwise complete. On the other

hand, an acute attack of the arthritic type is very rarely indeed fatal, and when this is the case death is usually due to the supervention of cerebral rheumatism and hyperpyrexia.

In thus sketching briefly the clinical symptoms of the arthritic type of the disease, I do not wish to imply that the more severe types of rheumatic fever with visceral inflammations are complications of the true disease. To this extent they are complications, that they introduce many new dangers, and need a more elaborate watchfulness and care in their treatment, but they are, I believe, essentially rheumatic manifestations and not the result of secondary infection.

To enter into detail upon these various manifestations would be to trespass upon the articles already devoted to their study, and it must suffice to point out the chief clinical symptoms which lead the physician to recognise their occurrence.

First in order of importance are the cardiac affections—endocarditis, pericarditis, and myocarditis. The proper understanding of these will be much simplified if it is recognised that in severe rheumatism of the heart all the structures are injured, and that the condition is one of carditis—but in some cases the brunt of the disease falls on the valves, in others on the pericardium, and again in others, probably, on the myocardium. Recalling the teaching of the morbid anatomy, we must dismiss from our minds the idea that myocardial changes necessarily spread from the pericardial inflammation, and recognise that they are a morbid result of the rheumatic poisons upon the muscular structure, as much as are the somewhat similar though more intense changes in diphtheria the result of the toxins of that disease.

As age advances the cardiac affections become less frequent, but dilatation of the heart, the earliest evidences of which have already been described, still remains frequent.

Endocarditis.—It is difficult to determine at the bedside when the valves are actually inflamed, for the onset of the valvulitis may be at any period in the disease, and the amount of general disturbance very slight. Reduplication of the second sound over the impulse, especially if with this there is a soft systolic murmur which can be traced toward the left axilla, points strongly to some damage of the segments of the mitral valve, and though the murmur may subsequently disappear, this persistent alteration in the character of the second sound betrays that the valve has been slightly damaged.

Any rise of temperature or increase in the rapidity of the pulse must be looked for as an additional proof of the occurrence of endocarditis.

Malignant endocarditis is fortunately not a common occurrence in rheumatic fever, though from investigations made with Dr. Alexander Paine, I am convinced that this result may be a manifestation of rheumatic fever, especially when the valves have been damaged by previous attacks of rheumatism. The onset of this fatal form of valvulitis is usually very gradual; persistent rapidity of the action of the heart, loud and often musical murmurs, irregular fever, infarction, anæmia, and emaciation are among the important indications. In some cases there is doubt as to the diagnosis, even to the very end of the illness, but, as a rule, with the progress of the disease the symptoms diverge more and more from those which are associated with simple rheumatic fever.

Pericarditis.—It is the virulent rheumatic infections, such as are more frequently seen in childhood, that give rise to pericarditis, and its occurrence is always a grave matter. But to separate rheumatic pericarditis from endocarditis and myocarditis, and to study it apart from them, is difficult and in some degree misleading. There are many cases of carditis in which pericardial inflammation cannot be detected or does not occur, which nevertheless damage the heart very seriously, and their importance is apt to be underrated because pericardial friction has not been heard. Overmuch of the actual damage to the heart in rheumatism has very probably been laid at the door of pericarditis and the adherent pericardium, which is so frequent as a sequel.

Pain over the heart, dyspnœa, a rise in the temperature, pallor of the face, mental anxiety, and even delirium are among the early signs of pericarditis. The pulse is rapid, often irregular, and the tension remarkably low. The pericardial friction sound is frequent, and it may not be out of place here to insist upon the fact that the greatly increased area of the cardiac dulness that is so common is far more often the result of cardiac dilatation than of a large pericardial effusion.

Myocarditis. — The myocardial changes in rheumatic fever are very difficult if not impossible to diagnose, though we may often have good reason for suspecting them. Insidious myocardial disease of rheumatic origin is also very possibly more important than is generally recognised, for it is undoubted that there are cases of rheumatic morbus cordis, the symptoms of which are far more urgent than the physical signs would lead us to expect, and which are some of them due to damage to the cardiac muscle.

All rheumatic affections of the heart may commence so unobtrusively that nothing but a routine and careful examination in every case of rheumatic fever can protect us from error. And when rheumatic fever has left its traces on the heart, and the patient is numbered as one of the great group of cases of morbus cordis, it is more often a fresh attack of rheumatism

that injures the compensation of this damaged heart than any direct mechanical overstrain.

Phlebitis and Venous Thrombosis.—Phlebitis and venous thrombosis are among the more unusual occurrences, though within the last four years I have met with six undoubted examples, which leads one to suppose it may be more common than is perhaps thought to be the case. French observers have frequently observed the condition, and state that phlebitis is most common in the lower extremities. The phlebitis in my experience has commenced in the large veins of the arms and neck, and all tributaries of the superior vena cava, including the upper part of the vein itself, may be occluded.

The diagnosis rests upon the occurrence of localised œdema of the arms, face, or chest, with tenderness along the damaged veins, dilatation of the collateral vessels, and sometimes the discovery of firm cords in the position of the veins. In four of the six cases that I have seen there was also pericarditis.

Changes in the Blood.—*Anæmia.*—During an attack of rheumatic fever there is a fall in the number of red blood corpuscles. The decrease is usually rapid, and in convalescence this loss may also be rapidly repaired. There are cases, however, in which, as Dr. A. E. Garrod has pointed out, anæmia may persist for a long while, and in which there is a fall in the hæmoglobin worth of the blood corpuscles. There is very often a moderate leucocytosis, and this in exceptional cases may be very marked, and the leucocytes contain diplococci. In severe cases of pericarditis the diplococci circulate in the general blood-stream, and can be isolated from the blood, but in the less severe cases it is doubtful if even with 10 c.c. of blood any constant results would be obtained.

Respiratory Affections.—*Pleurisy.*—Rheumatic pleurisy is frequent where there is pericarditis, and those parts of the pleuræ which overlap the pericardium are especially liable to be attacked. Pleuro-pericardial friction is perhaps more frequent in rheumatic fever than in any disease, for as a rule this inflammation is plastic. Pain in the side, a catch in the breath, and a pleural friction rub are usually present in rheumatic as in other forms of pleurisy. Rheumatic pleurisy may no doubt also occur apart from pericarditis, and it is an important question that yet remains to be solved, how many primary pleurisies which are non-suppurative are rheumatic and not tubercular in their origin.

Pneumonia.—Rheumatic pneumonia is frequent in the severe attacks of visceral rheumatism. The clinical features are in some respects remarkable. It is, as stated in the study of the morbid anatomy, essentially a bronchopneumonia, and when due allowance is made for the collapse of the left lung, which always occurs when the heart is much enlarged, it is doubtful whether, as has been maintained, it occurs more frequently on the left side. It may certainly be present on both sides. The physical signs are sometimes much more extensive than the lesions would lead one to suspect, for the actual pneumonic change is small. The temperature rises abruptly, but falls by lysis rather than by crisis, and the rapidity of the resolution is remarkable.

Pulmonary Œdema.—Pulmonary œdema is rare, and, as described by French writers, occurs suddenly and unexpectedly in the mild cases. Such instances as I have met with have occurred in the severe forms of cardiac rheumatism. Sharp crepitations appear over the upper lobes in front, and then, if the condition is to prove fatal, quickly spread over the entire lungs. There is great respiratory distress, but in the early stage recovery is possible, and the crepitations may rapidly disappear.

It is a very interesting point that this œdema is probably not of mechanical origin, for it commences, as does the œdema in renal disease, in the upper lobes, and is more marked in these lobes after death. In connection with the occurrence of this œdema, it may be advisable to consider the possibility that intolerance of salicylates may be a factor in the causation.

Nervous Symptoms.—*Chorea* in adult life is rare, though young women during pregnancy may suffer sometimes most severely; and those who have had a previous attack, or have suffered from other forms of rheumatic fever in earlier life, appear to be the most liable. In adults also a condition may be sometimes noticed, during an attack of rheumatic fever, which can hardly be termed chorea, but in which the voluntary movements are uncertain, sudden, and sometimes irregular. Finally, there are cases of chorea in early life which persist for many years, and which may be truly called chronic chorea.

Peripheral neuritis is an occasional event, and atrophy of the small muscles of the hand may sometimes reach an extreme degree. In some cases muscular atrophy and arthritis combined produce a condition very closely resembling acute rheumatoid arthritis.

Hyperpyrexia; Cerebral Rheumatism of the French Writers.—This is the most fatal event that occurs in an attack of rheumatic fever, but is fortunately rare.

The rapid rise of temperature to such a limit as 108°, or even higher, forms the most striking feature; but the cerebral symptoms are also very remarkable, and they may occur before the temperature has reached a great height. There can be little doubt that behind these conditions there is some underlying cause which accounts both for the cerebral symptoms and the hyperpyrexia. Headache, restlessness, and insomnia may be the early symptoms. Sometimes much urine is passed, and the pain in the joints may

disappear and sweating cease. Later, delirium, coma, muscular tremors, and involuntary evacuations occur, and death may follow general convulsions. The temperature may rise with extreme rapidity, or after maintaining a high level for some days suddenly rise beyond the limits of safety. The condition is always one of extreme danger, and one which requires prompt and decisive treatment.

Angina Faucium.—There is no characteristic sore throat, but the most common type is probably the one described by Fernet, and quoted by Dr. A. E. Garrod in his treatise on rheumatism: "A diffuse erythematous redness is seen to occupy the whole of the back of the throat, and some œdematous swelling of the mucous membrane is present, which is most marked about the uvula, which is swollen and elongated. The pharynx is moist and free from all exudation; one or both tonsils may present more or less considerable swelling." The throat is one path of infection, but it would be unwise to claim it as the only one; at the same time, if inflammation is to be looked upon as an effort of resistance, it may be well to point out the possibility of a virulent infection by this channel without the occurrence of pain and inflammation. Such an occurrence in other infections—puerperal fever, for example—is thoroughly recognised. The infection may be terribly virulent, but local changes practically absent.

Peritonitis.—When we consider the frequency of pericarditis and pleurisy, it is interesting to find the rarity of peritonitis; but though rare, the occurrence is undoubted, and with the other symptoms of this condition loud peritoneal friction may be occasionally heard.

Acute dilatation of the stomach may follow rheumatic fever, and the digestion is often enfeebled for some time after a prolonged attack.

Appendicitis has been traced to rheumatic fever, and among others Dr. Burney Yeo, Dr. Sutherland, and Dr. Beverley Robinson have supported this view. It is very difficult to trace a definite connection between these diseases, and the subject is the more difficult because there sometimes arises in connection with appendicitis a septic arthritis which may closely resemble the arthritis of rheumatism.

Renal Affections.—*Nephritis*, though not common, has been observed by competent authorities, and it is very probable that the kidneys are often damaged by repeated attacks of rheumatic fever, without that damage reaching the stage of an actual nephritis. Rheumatic fever may in this way assist in the production of that condition recognised in later life as "granular kidney."

Albuminuria sometimes occurs, and, if there is infarction, hæmaturia.

Hæmaturia may also occur without any evidence of infarction in some malignant types of the disease to which Professor Litten has especially directed attention.

Subcutaneous Nodules.—Subcutaneous nodules sometimes occur in adults over the bony prominences and along the sheaths of tendons. In some seasons these nodules are far more common than in others, and it is then they are most liable to be found in adults, and it is then also they may occur without severe cardiac disease, which, as Dr. W. B. Cheadle has insisted, is the usual accompaniment. Myalgia may be a troublesome symptom in an attack of rheumatic fever, when the joints in the proximity of the muscles show no signs of inflammation.

Cutaneous Manifestations.—Although much clinical information has been obtained upon the skin affections, there is still a good deal of obscurity around the subject, on account of the difficulty in determining which of these lesions are truly rheumatic.

Among the acute affections the erythemata and purpura are well recognised, and if some forms of psoriasis and scleroderma are accepted as rheumatic, these would represent skin affections of the more chronic type.

The erythemata are classified according to their characters as erythema, marginatum, papulatum, bullosum, and nodosum.

The first two are also included under the term erythema multiforme.

The marginate form appears in circles of various sizes, often called by the patient "ringworms," the dull pink colour of which quickly changes in the centre to a livid hue. The circles often coalesce, and thus are formed gyrate patterns which fade and leave a dull stain. In association with these forms of erythema, purpura is not uncommon, and occasionally a subcutaneous nodule appears in the centre of a patch.

In the papular forms the areas are raised, usually symmetrical, and often show a slight bruising. These eruptions, except for some smarting, give rise to little discomfort. In rare cases there is extensive purpura without the occurrence of hæmorrhages from the mucous surfaces, and sometimes large sanious bullæ may be formed. If the process be closely watched, erythema is found to precede this purpuric condition, but the change is rapid and very complete. When these stages are passed through very rapidly, and there is active rheumatism as well, that condition of purpura results which may be truly called rheumatic, but there are other forms of purpura associated with arthritis which it is difficult to feel confident are truly rheumatic.

Erythema nodosum is a remarkable affection, in which intensely tender nodes appear most usually over the shins, but also over the forearms, thighs, and elsewhere. At first they are a bright pink colour, later they are a dull red, and may pit on pressure, and finally they leave

for a while a bruise-like stain. In rare instances subcutaneous nodules occur which differ considerably in their distribution from rheumatic nodules, and over which the skin is not discoloured.

Whether erythema nodosum is rheumatic is still much disputed, and the question cannot be said to be definitely settled. At present it is perhaps the safest plan to make a compromise, and say some cases are rheumatic and some are not; for, on the one hand, the late Dr. Begbie, Dr. Stephen Mackenzie, Dr. Cheadle, and others have brought forward good evidence of such an occurrence in rheumatism. On the other hand, competent authorities have given good reasons to show that it may occur quite apart from rheumatic fever.

Dr. Goodhart and others have looked upon psoriasis as a rheumatic affection, and there seems excellent reason for this belief. Scleroderma is so rare that it is difficult to prove the association, but Dr. Ball, Sir Dyce Duckworth, Dr. Crocker, and others have brought forward clinical evidence in its favour.

Urticaria is believed by many to be a rheumatic manifestation, and Dr. A. E. Garrod has noted that in some cases a localised œdema may replace an urticarial or erythematous eruption.

It is not unlikely that the sudamina of rheumatic fever are rheumatic manifestations.

Exophthalmic Goitre.—Dr. Samuel West has drawn attention to the frequency with which a history of rheumatic fever is obtained in these cases. The association is a very remarkable one, of which I have seen several instances. In rare cases inflammation of the thyroid gland has been observed.

Conjunctivitis and iritis appear to be rare, and there is doubt whether the latter ever results from rheumatic fever. I would venture to throw in my opinion with those who believe there is a true rheumatic iritis.

The Diagnosis of Rheumatic Fever

When the diagnosis of rheumatic fever is considered, we are struck with the great change that has taken place in the views upon arthritis. The investigations of bacteriologists have demonstrated that arthritis is a result of many and various infections: the mention of tubercular, pneumonococcal, streptococcal, staphylococcal, and gonococcal arthritis in addition to the rheumatic form is sufficient to exemplify this, and in each case it is admitted that the arthritis is a manifestation of the disease, and not the disease itself. No doubt the arthritis in each instance often presents some peculiarity in its course, as does the disease which is the cause, but an attempt to found a diagnosis upon some minute detail of morbid anatomy is no longer relied upon as satisfactory.

It is to be hoped that some means will arise by which the diagnosis of rheumatic fever can be pushed, as it were, a step farther back. Those symptoms which we now look upon as only suspicious will then be recognised as certain indications, and there will result a greater likelihood of forestalling the cardiac complications. The infective view of the disease holds out a promise that this advance will be made, and although it is not likely that the morphological characters of the diplococcus will assist in the diagnosis, some peculiarity in the cultural characteristics may eventually prove of great service.

At present the difficulty in the early diagnosis is to separate rheumatic fever from other acute fevers, and in particular from scarlet fever, influenza, and enteric fever when there is arthritis early in the illness. Acute osteomyelitis may lead to a serious error. Again, when tonsillitis occurs it is difficult to say whether this is a local affection only, or a manifestation of the more serious disease. It may, indeed, be necessary to suspend judgment for a time, but if all the facts of the illness are carefully weighed the delay is usually short.

Later, when arthritis has already developed, a broad survey is taken of the entire case: the age, the family history, the character of the arthritis, the implication of the heart, the occurrence of subcutaneous nodules. All these and other points as well are studied, and then in most instances the diagnosis is easy.

Rheumatoid Arthritis.—In the present state of our knowledge of rheumatoid arthritis, it is difficult to lay down any sure rules for diagnosis, and it is quite probable that some of these cases are really examples of inveterate rheumatic arthritis. On the other hand, an insidious disease which, according to Mr. Jonathan Hutchinson, is most common in delicate women of gouty inheritance, in which intractable progressive arthritis, often curiously symmetrical, is accompanied by great muscular wasting, and in which functional rather than organic disease of the heart is the rule, points to that form of joint affection which is called rheumatoid arthritis. The weight of the disease falls in these cases upon the joints, the muscles, and the nerves, and, though it is a rule by no means without exception, the heart is spared the severe inflammations of rheumatic fever.

Rheumatic Fever and Gout.—I feel that to speak of rheumatic gout is to be guilty of something of a heresy, but presumably there can be no reason why the rheumatic infection should not attack a gouty person. If it be allowed that it is the tissue metabolism in gout which is peculiar, then it is highly probable that the rheumatic infection will be modified by the peculiar soil in which it has gained a foothold, and a condition will result which is rheumatism in a gouty subject. On the other hand, a patient who has suffered from rheumatism in early life may so live in his adult life as to

become gouty, and gout in a rheumatic subject may be also modified in its character. The tendency is for the arthritis in these cases to be unusually obstinate. In lead-workers who have suffered from undoubted rheumatic fever, a condition of arthritis in later life is seen which is called gout—a condition far more intractable than the usual rheumatic arthritis, yet not infrequently called gout, chiefly because the patients were workers in lead.

Pyœmic Arthritis.—In severe cases of suppurative arthritis, rigors, high irregular fever, delirium, and rapid prostration are usual. But there is a group of cases vaguely termed "septic" which do not run this severe course, and which, if we judged by arthritis only, would be very difficult to separate from rheumatic arthritis. Such cases require further elucidation. Again, there is a form of pyæmic arthritis met with in drunkards whose constitutions are shattered, in which the temperature does not range high, and may indeed even be subnormal for a time. The joints are swollen and tender; there are tremor and wandering of the mind at night. The pulse is feeble, and the extremities are cold. To mistake this condition for rheumatic fever is a serious error, for it is invariably fatal, and the end comes with great rapidity.

Gonorrhœal Arthritis.—The diagnosis in this case rests chiefly upon the occurrence of a urethral discharge, or a history of a recent attack of gonorrhœa. The heart is rarely affected, and conjunctivitis and iritis are much more frequent occurrences than in rheumatic fever.

Traumatic arthritis is usually monarticular, and there is a definite history of injury to serve as a guide, but it should be borne in mind that in rheumatic fever there may occur an arthritis of one joint only. Errors may then easily arise. The patient may say, "I have sprained my ankle," and his diagnosis be too readily accepted, when a more careful search would have discovered fever, and perhaps inflammation of the heart. Acute monarticular rheumatism of the right hip-joint may be mistaken for appendicitis.

The arthritis that follows dysentery and other ulcerations of the colon in the early stage resembles a pyæmic arthritis in its severity, and with it there may be extreme muscular wasting, nevertheless the recovery is often very remarkable. The diagnosis rests chiefly upon the history of the disease from which the illness has dated. With tubercular, syphilitic, and pneumonococcal arthritis, the same broad survey of all the facts must be taken in arriving at a conclusion.

Allusion may here be made to the value of the salicylate treatment as a diagnostic aid. The rapid way in which many cases of rheumatic polyarthritis subside under this treatment is an undoubted assistance in diagnosis, but it is far from certain that we are justified in making the result of this treatment a complete test of the rheumatic nature of a doubtful case. The action of salicylates is so little understood, and the nature of the rheumatic processes at present so mysterious, that it seems hazardous to build up a diagnosis on such evidence, however strongly the practical convenience of it may appeal to us.

The Prognosis of Rheumatic Fever

It is very rarely that a patient does not recover from a first attack of rheumatic fever, though hyperpyrexia occasionally supervenes and proves rapidly fatal, and in exceptional cases carditis has been the cause of death. In the vast majority of cases recovery occurs, but it is unfortunate that this recovery is often incomplete, and even more unfortunate that one attack does not protect in the future.

The prognostic indications of the various heart affections are dealt with in the articles devoted to those diseases. Here we are only concerned with the prognosis of acute rheumatism, for which only general rules can be given. The younger the patient, and the more pronounced the hereditary tendency, the greater fear there is of a future attack, and this fear will be enhanced if the circumstances are poor and the surroundings unhealthy. Repeated attacks of rheumatic fever in early life usually leave some organic heart disease behind them.

The occurrence of pericarditis, pleurisy, and pneumonia point to the virulent types of the disease, and subcutaneous nodules, as Dr. W. B. Cheadle has insisted, are additional proofs of this, although, as mentioned earlier in the article, there are some exceptional instances.

The supervention of the malignant type of endocarditis is extremely grave, and such cases seldom recover. The persistent types of subacute rheumatic fever, in which pericarditis and other manifestations appear from week to week and month to month, are of bad prognosis, and it would seem that when the health of the patient has been long undermined by the rheumatic processes and the mechanical difficulties of chronic valvulitis, a condition is reached in which active rheumatism smoulders on unaffected by any methods of treatment. On the other hand, many cases of articular rheumatism, especially in adults, recover completely, and, even if valvulitis has resulted, the mitral incompetence is often so slight as to produce no tangible injury to health, provided there are no further attacks of rheumatic fever. Pericarditis is justly dreaded, but there is good reason to suppose that recovery from this may sometimes be complete. As a rule the joints recover completely, and, even though pain and stiffness remain for some while afterwards, assiduous treatment will generally meet with success. There are exceptional cases in which deformity results and remains permanent. Such

cases, though they resemble rheumatic fever in their varied manifestations, are, in the character of the changes in the joint, more akin to rheumatoid arthritis.

The prognosis in hyperpyrexia is very grave, but the prompt treatment by cold baths has made it far more favourable than it used to be.

The Treatment of Rheumatic Fever

Although there is no method of treatment which is generally recognised as specific, it is a disease which skill and care can do much to alleviate. The tendency that there is to natural recovery makes it especially difficult to ascertain the amount of improvement that directly results from treatment by drugs, and as a result many different remedies have been used and later abandoned. There seems little doubt that some manifestations respond to treatment better than others, and that of these arthritis is the most, and carditis the least, amenable. Experience has shown also that depleting measures, such as free venesection and hard purging, are out of place in the management of rheumatic fever.

Prophylaxis.—It is only within recent years that the great importance of the early warnings of rheumatic fever have been insisted upon, and the prophylactic treatment can hardly yet be said to have developed. The search for suitable climates for rheumatic children, for the paths of infection, and for the methods by which infection may be avoided, will very probably lead to good results. And the influence of bad sanitation on the occurrence of rheumatic fever seems especially worthy of attention.

Treatment of an Attack of Rheumatic Fever.—*General Measures.*—Rest in bed is the remedy that at present stands first, and has been insisted upon by all the greatest teachers upon the treatment of the disease.

Strong nurses are required when the patient is an adult, for the pain on movement causes complete helplessness, and the ineffectual efforts of a nurse, however skilful, who is physically unable to lift the patient properly, is an additional cause of suffering.

The bed should be strong and firm, the height a convenient one, and the breadth not so great as to make the patient difficult to move from either side.

On account of the profuse sweating he should lie between blankets, and these can now be obtained of such softness as to be suitable for the more delicate skins.

Lastly, the bed-gown should be of flannel, and by means of suitable tapes should allow of an inflamed joint being easily reached. When the sweating is very profuse, occasional gentle drying with a soft warm towel is grateful, and helps to diminish the unpleasant odour which is so frequently noticed.

Local Treatment.—Hot dry wool is a very useful routine application, and plenty of this should be gently bandaged around the joints, or, if wool cannot be obtained, fine sand or salt baked until dry and placed in suitable bags make good alternatives.

Opium and belladonna liniment, warm alkaline lotions, and hot fomentations can be used, though the aim should be, as far as possible, to avoid frequently disturbing the limb.

When the pain in one joint is excessive, plaster splints are useful for keeping the limb absolutely at rest.

Cold applications are not so generally useful, and though blisters or a light touch with the cautery are of great service in chronic arthritis, these are not so suitable when many joints are affected with acute inflammation.

When the joints remain stiff, weak, and painful after an acute attack, gentle massage, dry heat, and hydropathy are indicated, and the compound mustard liniment, though unpleasant, is a useful aid in relieving pain when the more expensive methods are out of the question.

Diet.—The diet during an acute attack should be light and nourishing, milk diluted with a mineral water or barley water, light soups, and later farinaceous foods, given at regular intervals. Strong soups, and the many patent meat preparations, although they are stimulating, throw a strain upon the kidneys which in acute rheumatic fever is better avoided.

When the attack is less acute the diet need not be so rigid. It is difficult to explain the reasons for the strong objection there is to the use of animal foods in rheumatism, but physicians of wide experience have insisted that relapses are favoured by their use, and that they have had cause to regret a concession to the patient upon this point. Much probably depends upon the character of the attack, and there can be little doubt that in the protracted and asthenic type in which the temperature is but little raised, a generous diet is better than a restricted one.

If, on the other hand, in a strong adult the diet has been limited, the return to meat and ordinary meals must be gradual, for the digestion is considerably impaired by the disease. The use of alcohol is not required unless there are indications of cardiac failure, and the physician will then be guided by such indications as hold good in the treatment of diseases of the heart.

Treatment by Drugs.—*The Salts of Salicylic Acid and Salicin.*—These drugs are very widely used, especially in the treatment of the arthritic type of rheumatic fever.

First introduced by Dr. Maclagan into England, they have undoubtedly proved of great value, and have relieved an immense amount of suffering; under their influence the pain of the arthritis is rapidly eased, and the tempera-

ture falls, but the severe rheumatic affections, such as pericarditis and endocarditis, do not seem to respond in the same striking manner, and nodules may increase in size and number while full doses are being taken. Many believe the action of these salicylates is specific, but it appears certain that in severe rheumatic pericarditis this specific action cannot be clearly recognised when doses are given within the the limits of safety.

They may be used to arrest a threatened attack of rheumatic fever, to control the developed disease, and to prevent relapses.

It is in the earlier stages, before definite arthritis or endocarditis has developed, that we should be led to expect the best results from their use, but it is difficult to feel sure at such an early stage that the attack would have developed into the pronounced disease. There is, nevertheless, in this early stage good reason to suppose that they are of value. When the disease has fully developed, it is generally agreed that the drugs should be pushed in full doses for a short time to get the patient rapidly under their influence. Some authorities, having achieved this object, stop them abruptly; others gradually diminish the dose and increase the intervals between them. The latter course appears to be the more satisfactory. If, then, we suppose a strong adult suffering from an acute attack of arthritic rheumatism, the bowels should be opened by an aperient, and twenty-grain doses of salicylate of soda be given every two hours until the temperature falls and the pain is relieved, then the interval be increased to every four hours, and later the dose gradually diminished. When the temperature and arthritis have been subdued for about ten days salicylate of quinine can be given, and finally quinine in an effervescing alkaline draught be substituted.

Unfortunately there are some difficult questions that must be faced in the use of this method. The salicylates have been widely looked upon as depressant to the heart, and their efficacy in rheumatic carditis doubted. Should we, then, adopt this treatment in preference to others, when there is pericarditis or other clear indications of active and severe heart disease? It is, indeed, a momentous question, and one which, in the face of the antagonistic views of physicians of wide experience, may well be answered with hesitation.

Further, it must be allowed that the quality of these drugs has been improved of recent years, and that, as Dr. D. B. Lees has pointed out, some part of the depressant action which has been ascribed to them is really the result of the rheumatic poison upon the heart wall. But until more convincing evidence has been given of the specific action of these drugs in rheumatic carditis, it is, I believe, safer not to push them when there is active heart disease, but if there be distress from a coincident arthritis, to use them then in moderate doses to control the pain.

Another answer to the objection that they are depressant, may be that though this is true of their action upon the normal heart, such is not the case when the heart is affected by rheumatic fever, because their action is specific. It is difficult to answer this contention, for both the action of the disease and of the drugs—if they are depressant—tend to cause cardiac failure. Those, then, who favour their use are apt to ascribe acute cardiac failure to the disease, those who hold them to be depressant will attribute it to the drugs. It appears to me that they are depressant to the heart in active rheumatic affections, as they are to the healthy heart, and that their use in full doses in acute carditis is hazardous. Pallor of the face, dangerous vomiting, air hunger, and rapid cardiac failure are symptoms which, if they develop acutely in a case of rheumatic carditis treated by this method, would suggest to me the toxic effects of the drug rather than of the disease.

Those cases in which there is obstinate relapsing rheumatism of the joints are often benefited by the prolonged use of moderate doses of salicylates.

The particular preparation that will be given in any case differs with the practice of the individual prescriber. The most effectual, though more expensive drug, is the natural salt. A milder preparation, useful for delicate persons and children, is salicin, but a good sample of salicylate of soda will, in most cases, answer every purpose, and the taste is well covered by spirits of chloroform and tincture of cardamoms. There are exceptional cases in which moderate doses will cause delirium, deafness, and vertigo. A comparatively new preparation, acetyl-salicylic acid, or aspirin, has come into use, and is claimed to stimulate the heart and to avoid any nervous symptoms. It has yet to stand the test of experience. The drug may be prescribed with syrup of lemons in doses from 5 to 20 grains.

Before the treatment with salicylates was generally adopted, large doses of alkalies were believed to be of value, and this method of treatment still retains to some extent the confidence of the profession. If they are not used alone they are often combined with the salts of salicylic acid. The citrates, bicarbonates, and acetates of potash are the most frequently administered, and when this method is pushed, half a drachm of bicarbonate of potash and the same of the citrate are given in an ounce of water, every four hours, until the urine is alkaline. When this has been done, the urine is kept slightly alkaline by gradually diminishing the amount of these drugs, and the treatment is persisted in until

the symptoms abate. Later, quinine may be added. The effect of this treatment must be carefully watched, for it may prove too depressant if the circulation is feeble, and with some patients these large doses of alkalies cause severe diarrhœa.

The view that rheumatic fever is the result of an infection brings into prominence the importance of the constitutional resistance. This factor is a very important one in this disease, and one of which there is danger of losing sight, if exclusive attention is directed to the effect of the disease. Energetic and routine treatment by powerful drugs may damage this resisting power of the individual more than they influence the disease. With opportunities of studying different methods of treatment, it has appeared to me that in the dangerous and complicated cases of visceral rheumatic fever the best results are obtained by palliative measures; rest, careful feeding and nursing, moderate doses of salicylates for pain and arthritis, stimulants for cardiac failure, small doses of opium for cardiac distress and insomnia, mild tonics for convalescence—in brief, a watchful but gentle treatment. Under such measures some of the most desperate cases have rallied, and fewer, I believe, have gone to the bad with the sudden development of urgent symptoms.

Since there is so much evidence to show that the tonsils may be a site of infection, the treatment of the throat is important, and will be carried out on general principles. A gargle containing salicylate of soda may be used, or glycerine of carbolic acid, or formalin, and some have advocated guaiacol as especially valuable. In spite of this treatment of the throat, the diplococci in the other local lesions may doubtless multiply, and no treatment of the throat alone can suffice.

Repeated attacks of tonsillitis may result in a permanent enlargement for which tonsillotomy may be needed. The operation, though needful for the general health, will not prevent further attacks of rheumatic fever, and on several occasions to my knowledge an attack has rapidly followed this operation, though presumably these were coincidences.

Otitis media, with which an attack of rheumatic fever may sometimes begin, must be treated on the usual lines.

Treatment of Hyperpyrexia.—No time should be lost in giving antipyretic drugs, but the patient should be placed in a bath at the temperature of 65° with ice applied to the head, and the water rapidly cooled by adding blocks of ice. If the temperature of the patient is, for example, 107°, when it has dropped to 102° he should be taken out, rapidly dried, and wrapped in a blanket. As a rule the temperature will continue to fall for a while, but should it rise again this treatment must be repeated. It is a severe remedy for a desperate condition, and throughout the time of immersion the patient must be most closely watched, and be removed if there are signs of collapse, and stimulants freely administered. If the temperature should fall below normal after removal, hot bottles must be placed round outside the blanket, and stimulants given. Ice-packing is valuable when a bath cannot be managed. A sheet is wrung out in iced water, the patient swathed in it, and then rubbed with blocks of ice. The effect must be closely watched, for it is a drastic remedy.

When convalescence has been established, the question arises as to the length of time complete rest should be insisted upon. Much must depend upon the condition of the heart and the character of the attack. The guiding rule is caution, for premature exertion appears to be especially liable to cause a relapse. On the whole, it is better to be free from any arbitrary time limit, for this introduces the danger that when this time is past the patient will throw caution to the winds. If the pulse, the temperature, and the threatenings of active rheumatic manifestations are taken as guides, the return to an active life can be gradually attained, and every forward step carefully tested. The tedious nature of the illness makes the patient chafe at the enforced rest, and considerable tact in the graduation of the amount of physical exertion is required from the medical attendant.

The opposite danger, that of prolonging too much the period of rest, is also a real one. An invalid life is fostered, and on the young to whom movement is a natural tonic needless confinement is positively harmful.

When recovery is completed, and if expense is no object, it is well for the patient to leave England during the next year for a dry, warm, and equable climate during the autumn, winter, and early spring; and if a child, great care, especially during the autumn, will be necessary for some years to come. Warm clothing, warm socks, and thick boots are very necessary for rheumatic subjects in cold and damp weather, and woollen garments should be worn all the year round.

There remains the question of tonics. Strong preparations of iron, although there may be considerable anæmia, are not well borne. Quinine and arsenic in alkaline solutions, the milder preparations of iron, or the time-honoured prescription of ammonia and bark, are more useful.

An Antitoxin Treatment.—It is a natural hope that in future some antitoxin may be found which will be serviceable in treatment, and already antistreptococcic sera have been used. Unfortunately, treatment by antitoxins, except in diphtheria, have not yet met with wide success, and rheumatic fever with its numerous local lesions differs widely from that disease. Further,

the greatest caution will be needed in judging of the value of such treatment, for, on the one hand, rheumatic fever naturally tends to recovery, and, on the other, the various members of the great group of micrococcal infections are not yet clearly differentiated from one another. But it would be unwise to prophesy on this matter.

In conclusion, I am indebted to my colleague, Dr. A. Paine, Bacteriologist to St. Mary's Hospital, London, for permission to make free use of our joint investigations upon which the details of the bacteriology and histology in this article are based, and which have enabled me to treat confidently of rheumatic fever as an infective disease.

Rheumatism in Children

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There is a considerable difference in the manifestations of rheumatism as ordinarily seen in children and those occurring in the same complaint in adults. In both there is the same difficulty in defining what symptoms may or may not with any degree of precision be classified as rheumatic, and the younger the patient the more this difficulty is enhanced. In what is known as acute rheumatism, or rheumatic fever, in adults, with the high temperature, the profuse, sour-smelling perspiration, and the swollen and acutely painful joints, we have a complaint with well-defined symptoms, admitting of but little doubt in their interpretation or diagnosis. Such a combination of symptoms is very rarely seen in young children, and it is only towards the age of puberty that rheumatism in them takes on the adult type. In the complaint, as ordinarily seen in children, the temperature seldom rises above 101° unless definite tonsillitis or pericarditis be present; the skin, instead of moist, is usually dry; pain s generally much slighter than in adults, and if there be any recognisable arthritis, it is slight in degree, and very rarely accompanied by any redness of the affected joints. Such terms as acute and subacute have but little significance or propriety when applied to rheumatism in childhood. Distinguishing features of rheumatism in children, as compared with the disease in adults, are usually much slighter degrees of arthritis, the greater tendency to the occurrence of skin eruptions and subcutaneous fibrous nodules, and the much greater frequency of cardiac complications, and this last in spite of the often seemingly trivial character of the preceding or accompanying symptoms. There is also a tendency for some one single symptom to assume such prominence that the others are apt to be obscured. Stress must be laid upon the disconnected and apparently trivial outward phenomena of the complaint, as a correct diagnosis is all-important, having due regard to the frequency and gravity of the associated cardiac complications.

PATHOLOGY.—This is the same, undoubtedly, as that of the complaint in adults, and rests on the same obscure basis. The theory that the disease is due to lactic acid in the blood has little or nothing to support it. The malarial origin of rheumatism, based mainly on the effects of the salicylates in controlling it, can hardly now be advanced in the face of modern discoveries regarding malaria. For the view, once widely held, that rheumatism was dependent upon some impression on the nervous system there is still much to be said, although the arguments adduced in favour of it never advanced beyond pure hypotheses and suggestions. At present the theory that rheumatism is of bacterial origin is the one most in vogue, and various forms of micro-organisms, chiefly streptococci, have been isolated from the blood and other constituents of the body from sufferers with it, and the claim is put forward that they are the cause of the disease. The uniform relation often obtaining between the development of arthritis and crops of subcutaneous nodules and a previous sudden rise of temperature lend further support to the bacterial theory. Allowing all this, however, and acknowledging the excellence and importance of the recent work of Poynton and Paine, it cannot be claimed that the bacterial origin of rheumatism is conclusively proven. Their experiments require repetition and confirmation by themselves and others before being accepted as definitely conclusive. Many of the pathological changes produced in the endocardium and other structures of rabbits through the agency of the special diplococci, which they regard as one of the existing causes of rheumatism, have been asserted by equally competent observers to occur in the same animals by means of

several other micro-organisms. Moreover, it is difficult to explain the sudden onset of certain manifestations, presumably rheumatic in character, that come on in persons subject to rheumatism, either directly after, or indeed simultaneously with, the exposure to cold or damp, on the assumption that the complaint is one of bacterial origin. Where all is so unsettled there is no need to discuss the question at any length, more especially as it is treated of elsewhere in this volume (p. 579 *et seq.*).

Etiology.—Inheritance from father or mother is a powerful disposing cause, and this is largely increased when both parents are rheumatic. When one member of a family, too, has suffered from rheumatism, there is a much greater liability for the brothers and sisters to incur the complaint than the members of other families, even when there is no parental inheritance. That living in cold, damp, low-lying situations predisposes to rheumatism is beyond question, and of late it has been stated that dry hilly places exposed to bleak winds are equally unfavourable as regards the complaint. Males suffer more often than females owing to more frequent exposure to wet and damp, but apart from this, either sex under the age of ten is equally liable to the complaint. Between the ages of ten and fifteen, however, there is a marked preponderance of cases amongst girls, and this has been put forward as some explanation of the greater prevalence of mitral stenosis in the female sex. In temperate climates the disease is most prevalent in the spring. There is some evidence for believing that red-haired children are more prone to rheumatism than others. A very powerful factor in predisposing to the complaint is the fact of having suffered from a previous attack, there being no doubt that subsequent attacks are incurred with much slighter causes than those that occasioned the primary one. Immediate exciting causes are exposure to prolonged damp and undue physical fatigue, a combination of these two, indeed, accounting for considerably more than half the total number of cases.

Age. — Probably no age is exempt from rheumatism. Cases with unmistakable outward signs in the joints, etc., are exceedingly rare under the age of two years, and rare under that of three years. After the latter age attacks occur more frequently with each year of life up to the age of ten years. Possibly more attacks occur during the second decade of life than any other, and it is uncommon for a primary attack to occur after the age of twenty. Owing to the slight character of the symptoms in infancy it is probable that the frequency of rheumatism at that age is underestimated, and it is not uncommon to see little children with cardiac lesions of some standing, presumably rheumatic in origin, but where the outward symptoms of that complaint had been so trivial at the times of attack as to be wholly overlooked.

Onset.—In older children this may be more or less sudden, with marked pyrexia and acute pains in the joints. Such an onset is very seldom present, however, in the younger subjects. In these there is almost invariably a history of indefinite pains in the joints, the knees in most instances at the start, and it is only after these pains have lasted several days, or even weeks, that an increase of them, or swelling of the joints, calls attention to the true nature of the attack, and compels the confinement of the child to bed. Rigors and other nervous phenomena that occasionally usher in the disease in adults are practically unknown in children under the age of puberty.

Symptoms. — The isolated character of the symptoms in the majority of attacks in children, in contrast to the complex grouping of them that mark the more definite attacks in adults, justifies the plan proposed of dealing with each of them in detail. It is only in children, perhaps, that a confident diagnosis of rheumatism can be made from the presence of such isolated symptoms.

Pain.—This is almost invariably present, at some time or another, in every attack. In many cases, indeed, it is the only symptom complained of. It varies much in intensity and duration, but in both these particulars it seldom reaches the same height in children as in adults. It cannot be too carefully remembered that, in the vast majority of instances, the so-called "growing pains" of children are rheumatic in nature. It is far from uncommon to find advanced endocarditis in children in whom the only symptom of rheumatism has been these "growing pains." The history of "growing pains," too, often settles the rheumatic origin of many cases of chorea in the absence of other symptoms. When pain alone is complained of, the greatest care is necessary in ascertaining that it is limited to the joints before deciding that it is due to rheumatism, as in many febrile and other diseases children complain of vague pains in other parts of the limbs than the joints. The effects of passive movement may assist in this inquiry, but, for obvious reasons, cannot be definitely relied upon. When arthritis is present the pain is generally greater, and, indeed, it may be said in general terms that the amount and duration of the pain is directly proportionate to the extent of the arthritis. In some cases the pain is not limited to the swollen joints, but extends to the tendons of the muscles entering into their formation, and very exceptionally it can be unmistakably elicited by pressure over the nerves of the limbs away from the neighbourhood of the joints. Pain in the pericardium generally, but by no means invariably, attends the onset of pericarditis. Præcordial pain, too, may accompany

the beginning of endocarditis, but more usually this occurs without any such accompaniment, and if any sensation be complained of in the region of the heart it is generally one of vague uneasiness rather than actual pain. Not very unfrequently pain in the region of the neck, causing "stiff neck," is the only outward sign of rheumatism, and this pain may occasion contracture of the muscles on one side (rheumatic torticollis). As many other complaints may cause "stiff neck," or torticollis, these last will have to be excluded before the diagnosis of rheumatism is entertained.

Sweating.—The profuse, sour-smelling perspirations of adult acute rheumatism are only occasionally observed in children. In these last, as a rule, the skin is dry instead of being moist. Any sweating that occurs is usually late in the complaint, and is perhaps due to the action of the salicylates rather than to some intrinsic character of the disease. In the intervals between the attacks, however, rheumatic children are very prone to profuse perspirations on the slightest exertion.

Arthritis.—As a rule, this is less severe and more transient in children than in adults. In both there is the same tendency to symmetry, and for the swelling to disappear from points earlier attacked to involve others. The knees, ankles, and wrists, in the order given, are the joints most often attacked. Not very unfrequently the metacarpal phalangeal joints are involved with the larger ones, and much less frequently the corresponding joints in the feet. Cases in which the smaller joints are involved are generally severe. The swelling of the joints concerned is seldom of any great extent, and it is only exceptionally that there is any redness of the adjacent skin. The amount and extent of the arthritis is a measure of the severity of the attack, and it is very seldom that rheumatism of such severity as to cause definite arthritis is not attended with pronounced cardiac lesions in children under seven years old. In children under that age, too, with a history of past arthritis, there is strong presumptive evidence that the joint affection was not rheumatic if the heart be free. The swelling seldom remains in any one joint for many days, but accurate knowledge of the duration is rendered uncertain by the efficacy of the salicylates in removing the external signs of the complaint. If the swelling persist in spite of treatment, and is limited to one or perhaps two of the larger joints, then a suspicion of gonorrhœa should always arise, as cases of gonorrhœal arthritis are by no means unknown in even little boys and girls. In children a chronic synovitis is never left as a consequence of rheumatism, and contractures and other deformities of the joints are perhaps unknown. Very occasionally the arthritis is confined to one joint, usually the hip, and it is not uncommon to find these mon-articular cases mistaken at first for tuberculous disease of the affected joint. Some degree of arthritis, or pains in the joints, is a feature of the complaint in by far the greater number of cases. The interest and importance attaching to the mon-articular cases have, perhaps, led to an exaggerated estimate of their frequency on the part of certain writers. But it is in the diminished severity of the arthritis, and not mainly in its diminished frequency, that one of the chief distinctions between the rheumatism of childhood and that of adult age lies.

Pyrexia.—This plays a much less conspicuous part in the rheumatism of children than in that of adults. In contrast with what occurs in other complaints, in rheumatism the younger the child the less the tendency for the temperature to be raised. In slighter cases, marked by joint pain only with no accompanying arthritis, the temperature may not be raised at all, and this in many instances where the effects upon the heart are disastrous in the extreme. On the contrary, again, fever is never the sole manifestation of rheumatism in children, any increase of temperature being invariably accompanied by pain or some other more definite sign of the complaint. In ordinary cases, with an average amount of arthritis, the temperature seldom ranges above 100° or 101° Fahr., unless pericarditis or severe tonsillitis be present. With the onset of pericarditis the temperature may rise as high as 105° or 106°, and even higher still in cases where there is a combination of chorea with active rheumatism, manifested by such symptoms as pain and swelling of the joints. In cases such as the last pericarditis is almost invariably present as well. It is only in cases where pericarditis is present that we have in children any equivalent of the so-called "cerebral rheumatism" of adults, where the life of the patient is endangered from the pyrexia itself, independently of any other conditions. Much stress has been laid by some writers on the pyrexia consequent on endocarditis, but it can be safely said that any persistent temperature directly due to endocarditis is practically unknown with rheumatism in children. Any pyrexia that occurs is almost certainly due to the rheumatic symptoms that initiate the attack, and independent, probably, of any endocarditis that may ensue. Cases of endocarditis with persistent heightened temperature, where other sources of pyrexia can be excluded, should raise the same suspicion of malignancy in children as in adults, as although ulcerative endocarditis is rare in children, it is certainly far from unknown. An interesting phase of rheumatic pyrexia is afforded by cases where, after generally a sharp initial attack of fever, the temperature is persistently raised at some period of the day for many months, or even perhaps years. In such cases the tem-

perature may daily reach 104° Fahr., but beyond causing occasional feelings of discomfort when high, the continued pyrexia seems in no way detrimental to the child. The only explanation of such a pyrexia would seem to be that here the rheumatic poison, whatever it may be, has made a profound impression upon the cerebral centres, and the delicate heat-regulating machinery has been thrown out of gear. Treatment has little or no effect upon the disorder, and time alone brings about the restoration of the calorific balance.

Tonsillitis is a very frequent symptom of rheumatism in children, especially at the onset of an attack. The frequency with which it occurs before any other symptom has led to the suggestion from the upholders of the microbic origin of the complaint, that the tonsils are often possibly the points of entrance of the poison into the system. The tonsillitis is seldom of any great severity, and a suppurative rheumatic tonsillitis is exceedingly rare in children. The temperature bears some relation to the degree of tonsillitis, being generally higher the more the latter is marked. In very exceptional instances cases of endocarditis have been noted where the only evidence of rheumatism has been tonsillitis. This should lead to a careful and repeated examination of the heart in all cases of tonsillitis of uncertain origin. There is nothing distinctive, however, in the outward appearances of rheumatic tonsillitis, and a case can only be pronounced rheumatic with any precision when other evidences of rheumatism are present. Besides ushering in acute attacks, tonsillitis often occurs in rheumatic children in the intervals between them. Such tonsillitis is often accompanied by slight anæmia and sweating on the slightest exertion. Such symptoms should be regarded as danger signals, and lead to a strict supervision of the child. It may well be, too, that repeated tonsillitis is evidence of the rheumatic diathesis, where there has been a total absence of the more definite signs of the complaint.

Pneumonia.—This is so infrequent in association with recent and active signs of rheumatism in children, as to render it doubtful if its occurrence has any direct causal relation with the rheumatic process. In the later stages of heart disease, however, it is not uncommon to find signs of consolidation, along with pleurisy, at the left base of the lung. The symptoms and course of such consolidations are generally different from those of a typical pneumonia, and are usually preceded by the signs of pulmonary congestion and œdema in the affected lung.

Pleurisy. — In contra-distinction to pneumonia, pleurisy is very common in the rheumatism of children, and may be the only evidence of the complaint. In exceptional instances it is the result of the direct extension of inflammation from the pericardium. The usual signs of it are pain over the lower ribs, and not unfrequently a pleural rub can be heard on ausculation. When directly due to the rheumatic process it is generally of the dry and plastic variety, and is very seldom accompanied by any noticeable amount of fluid in the pleural sac. Where it occurs, however, in the later stages of heart disease, along with consolidation of the lung, the amount of fluid poured out into the pleura may be considerable, and aspiration is often necessary to relieve the enfeebled and failing heart. An overwhelming proportion of such cases occur on the left side, but a satisfactory reason for this is still to be found.

Peritonitis.—It cannot be claimed that the connection between rheumatism and any form of peritonitis has been established with any exactitude. Any case where fibrous nodules have been discovered in the peritoneum, as with the pleura and pericardium, has yet to be noted. At the present day there is a tendency, perhaps a growing one, on the part of certain authorities to ascribe some cases of appendicitis to a rheumatic origin. But for appendicitis to develop in the course of acute rheumatism, or for rheumatic symptoms to develop during an attack of appendicitis, must be events of extreme rarity, and such cases are certainly not greater in number than mere coincidence could account for. The only evidence usually forthcoming for associating such cases of appendicitis with a rheumatic origin is the effect produced by salicylates upon the course of the abdominal complaint. These effects are mainly shown in the reduction of temperature, and not in the direction of any curtailing the course of the disease. But salicylates influence the temperature in many diseases apart from those of rheumatic origin, and until further evidence is adducible beyond that furnished by their administration, the connection between appendicitis and rheumatism must be held to exist on an extremely feeble basis.

Nervous Affections.—So-called "cerebral rheumatism" or "rheumatic hyperpyrexia" is almost unknown in children, and the reality of a rheumatic meningitis, on which the hyperpyrexia was formerly supposed to depend, has yet to be proven by the publication of a single authentic case. Rheumatic children are very subject to headaches. These are often associated with anæmia, but in some cases Dr. Still thinks the headaches have a more direct relation to the rheumatic poison. Goodhart, Graham Little, and several other authorities find a strong connection between night-terrors and rheumatism, and Dr. Still not only agrees with this, but thinks day-terrors, somnambulism, somniloquence, habit-spasm, and lienteric diarrhœa, are often associated with the same complaint. All these last disorders have probably

no direct causal origin in the rheumatic poison, as cases of any one of them occurring along with active rheumatic symptoms are events of exceptional rarity. An explanation of the association of such nervous disorders and rheumatism is rather to be found in the "nervous" temperament, which is one of the most striking features in rheumatic children. As Dr. Still writes, "The nervous child is par excellence the nervous child." This is shown in some instances by an almost uncontrollable excitement over games and amusements, in some by excessive timidity, and in others by a sensitiveness or shyness which is almost morbid. One nervous disorder directly due to active rheumatism is peripheral neuritis, as evidenced by pain in the course of the nerves and subsequent wasting of the connected muscles. This is not so very uncommon, and would perhaps be more often found if systematically looked for.

Anæmia.—A profound degree of anæmia is very rapidly attained in the severe rheumatic attacks, and this may be very slowly recovered from. Along with the anæmia there is often considerable leucocytosis in the blood. In the intervals between more definite seizures, rheumatic children are prone to attacks of anæmia without any ascertainable cause. Chronic anæmia in rheumatic children is perhaps more directly ascribable to the frequently associated heart disease, but it becomes intensified with the onset of fresh symptoms of the complaint.

Wasting.—In the course of an attack of rheumatism there is often a rapid and marked loss of flesh, much greater than the pyrexia and duration of the illness would account for. It is true, too, perhaps, that thin children are more subject to rheumatism than those who are plump.

Subcutaneous Nodules.—Where so many of the other symptoms are inconspicuous and short-lived, too much importance cannot be placed upon the presence of subcutaneous fibrous nodules. In their typical forms and situations they are absolutely diagnostic of rheumatism, although it is quite possible that subcutaneous nodules may occur in syphilis, rheumatoid arthritis, and perhaps other complaints. Rheumatic fibrous nodules are small excrescences under the skin, varying in size from that of a pin's-head to that of about half a hazel-nut. They may be solitary or numerous, but most often occur in crops, with a tendency to symmetry in their distribution. Very frequently their eruption is preceded by a rise of temperature three or four days beforehand. They are strictly subcutaneous, and the overlying skin is freely movable over them. As a rule they are movable over the underlying structures, but in places where the skin is closely adjacent to bone, as on the scalp and the neighbourhood of the patella, it may be difficult or impossible to separate the nodules from the adjoining bone. When very small, they can often be seen better than felt, by putting the overlying structures on the stretch. Their far most frequent situation is the back of the elbow joint. Very often it is the only one in which they are to be found, and cases in which they occur in other places without the neighbourhood of the elbow joint being affected must be exceptional in the extreme. For their detection, then, in the vast majority of instances, it is sufficient to flex the elbow joint and carefully examine the structures on the posterior surface. Subcutaneous fibrous nodules occur in many other regions than the neighbourhood of the elbow joint. They are not infrequent about the patella, the iliac crests, the pericranium, the spine and margins of the scapula, the skin over the spinous processes of the vertebræ, over the bony prominences of the hands and feet, and elsewhere. Besides these nodules in the subcutaneous tissues, nodular thickenings, homologous in nature with the last, are apt to occur in the tendons of the muscles, more especially in the flexor tendons of the wrists, the tendons about the malleoli of the feet, and the flexor and extensor tendons of the fingers and toes. Occasionally they are found in the tendinous expansions of the abdominal muscles. They are painless in themselves, but are sometimes tender to pressure. Their presence in the scalp is often only detected by the comb catching on them when the hair is being dressed. Some vanish in the course of a few days, others persist for months, or even more than a year. Persistent nodules are often, or generally, accompanied by progressive endocarditis. Numerous nodules, and still more so nodules of large size, are omens of bad prognosis, and this out of all proportion to the extent any accompanying endocarditis may have attained. Subcutaneous fibrous nodules are invariably associated with cardiac disease, cases where they have been noted and there has been no demonstrable cardiac lesion being so exceedingly exceptional that they may be left out of account in dealing with the connection between nodules and morbus cardis. In cases where the heart has been healthy, the advent of subcutaneous nodules is almost invariably attended by the signs of recent endocarditis or pericarditis; and in cases where the heart is already affected, the onset of nodules is often accompanied by fresh additions to the heart symptoms, or an aggravation of those already existing. Considerations such as these lend strong support to the contention of Barlow and Warner that bodies analogous to the fibrous nodules in the subcutaneous tissues form the basis of the vegetations in the cardiac valves in rheumatic morbus cardis. Further support of this view is afforded by the fact that bodies histologically identical with subcutaneous fibrous nodules have occasionally been discovered post-

mortem in the cardiac valves. Not very infrequently fibrous nodules have been discovered in the thickened pericardium when the patient has suffered from rheumatic pericarditis during life. Much less frequently they have been found in the pleura and the fringes of the joints in cases of rheumatic pleurisy or arthritis.

Histologically these subcutaneous nodules are found to consist of fibrous tissue with some round or spindle-shaped cells, dilated blood-vessels, and a nearly homologous, more or less fibrillated "ground substance." This homogeneous substance has been shown to be a fibrinous exudation. These fibrous nodules are then, probably, the result of a local rheumatic inflammation. The invariable occurrence of other inflammatory manifestations simultaneously with that of subcutaneous nodules suggests that similar foci of inflammation to those occurring in the subcutaneous tissues may occur in the joints, the endocardium, pericardium, and the pleuræ. In these last situations the foci of irritation not only lead to the special form of inflammation attended by nodule formation, but to the ordinary forms of inflammation attended by serous effusion. Thus rheumatic arthritis, pleurisy, endocarditis, and pericarditis may originate in the same process that leads to the formation of subcutaneous fibrous nodules. It is their envanescent nature, probably, that prevents the more frequent discovery of these fibrous nodules post-mortem, not only in the subcutaneous tissues, but in other parts of the body.

As a rule the nodules disappear by a process of gradual resolution, getting smaller and smaller before their final disappearance. In exceptional instances this process is much hastened, and nodules of considerable size may disappear entirely in the course of a few days. As in the subcutaneous tissue, the nodules in the cardiac valves may disappear, and thus a cure be wrought occasionally in cases of organic cardiac disease. The hope afforded of such a chance is but a slender one. The shrinking and puckering of the delicate valvular structures secondarily to the inflammation attending the nodular eruption generally remains, however completely the nodules themselves may have resolved. Sometimes, instead of undergoing resolution, the nodules apparently become converted into fatty tissue. Over the point of the elbow it is not very uncommon for nodules to be succeeded by soft masses, like a small collection of fat, which persist long after the nodules elsewhere have disappeared. These apparently fatty tumours are generally about the size of a half hazel-nut, and the skin over them is often the site of an erythematous blush. Whether these soft masses are really fatty in character is as yet a matter of doubt, and hitherto their presence would seem to have escaped the notice of observers.

Occasionally the inflammatory process giving rise to subcutaneous nodules extends to adjacent periosteal structures, and periosteal nodes develop on the underlying bones. These rheumatic nodes have hitherto been only noticed when nodules are present, and generally the bony swelling is surmounted at first by one or more subcutaneous nodules. They usually persist much longer than the subcutaneous nodules, but have a tendency to gradually disappear in the course of time. The commonest site for such nodes is the subcutaneous surface of the ulna, and the nodes here generally have their long diameter in the direction of that of the limb. Still larger nodes, up to the size of half a walnut, may appear upon the skull, where the overlying scalp has been the site of subcutaneous nodules; less frequently than on the ulna and the scalp, rheumatic nodes develop on the surface and edges of the patella. They have been noticed, too, on the subcutaneous surface of the tibia, but are probably much rarer here than in the other mentioned places, from the fact that subcutaneous nodules very seldom occur over the surface of the tibiæ. It is quite conceivable, too, that rheumatic periostitis may occasionally arise, in places not amenable to inspection, from a primary form of inflammation in the periosteum itself, independently of any direct extension of inflammation from adjacent subcutaneous tissues. It is not very uncommon in cases where fibrous nodules are present, especially nodular thickenings in the tendons, for ganglia to develop about the wrists. Such ganglia are doubtlessly due to an inflammation of the sheaths of the tendons. Very occasionally several nodules become welded together by inflammatory exudation, and move together as one mass under the adjacent skin.

Fibrous nodules are undoubtedly most common in cases where arthritis and other of the more definite signs of rheumatism have been present. They have occasionally been found in cases of chorea and stiff neck, and certain skin eruptions where careful inquiry has totally failed to elicit any history of previous joint affection. Such cases are, however, exceedingly rare, and their number has perhaps been over-estimated by too enthusiastic contenders for the pan-rheumatic origin of chorea.

There is no single phase of rheumatism in children in which subcutaneous fibrous nodules have not occasionally been found. They are far most frequent in cases where definite arthritis is present, and Dr. Still found nodules in nearly 50 per cent of cases of articular rheumatism in children. They are next most frequent, perhaps, in cases where joint pains have been a prominent feature, but where the joint affection has not been of sufficient severity to lead to articular swelling or redness. In all cases of unmistak-

able rheumatism in children, nodules will be found in the early stages of the attacks in from 20 to 30 per cent of cases if systematically looked for.

Skin Affections. — These are comparatively rare, but are relatively much more frequent with rheumatism in children than in adults. Sudamina and miliaria have no direct association with rheumatism, and are perhaps less seldom seen in that complaint than in others where there has been pyrexia. The eruptions most commonly seen are various forms of erythema, and these may appear simultaneously with other rheumatic manifestations, or independently of them. Sometimes the erythema takes the form of small papular spots, widely scattered over the face and body generally. More commonly it appears as erythema marginatum, or erythema circinatum, and is most marked in the neighbourhood of the joints. The various forms of erythema are most frequent when nodules are present, and, like the last, perhaps afford some test of the intensity of the rheumatic affection. Erythema nodosum, although it appears independently of rheumatism, and runs a definite pyrexial course, is certainly more common in rheumatic children than in others. It is significant, moreover, of the connection between the two complaints that endocarditis occasionally ensues in cases of erythema nodosum where no other evidence of rheumatism can be elicited. Purpura rheumatica stands in the same relation to rheumatism as erythema nodosum, in that it runs a fairly definite course, is more common in rheumatic sufferers than in others, and is occasionally associated with endocarditis. Apart from purpura rheumatica other forms of purpura occur in rheumatism. Thus purpura may follow on intense erythema, or it may be primary in its manifestation, of rapid development, and attended by hæmaturia. Not very uncommonly a red rash, simulating the early stage of the scarlet fever exanthem, occurs in the febrile state of rheumatism, and may lead to some difficulty in diagnosis.

Endocarditis. — This is the most important feature of rheumatism, and the frequency with which it occurs is one of the most striking characteristics of the complaint in children. Donkin, in 70 cases of rheumatism, in children aged to fourteen, found heart disease in 87 per cent, and Church found cardiac affections in 83 per cent in children under the age of ten. According to Dawson Williams, the heart is affected in three-fourths of all the cases of acute rheumatism under the age of fifteen. The younger the patient and the more definite the signs of rheumatism, the greater the tendency for the heart to suffer. In children under the age of seven, with rheumatism of such severity as to cause any definite arthritis, it is a rare exception for the heart to escape. The presence of nodules, too, few or many, almost infallibly points to heart implication. The valve most frequently affected is the mitral, the aortic suffering much less frequently, and generally at a later stage than the mitral. Endocarditis may be associated with a slight rise of temperature remaining up after the disappearance of the outward rheumatic symptoms. Usually, however, it gives rise to no pyrexia, and is only revealed by physical examination, a procedure that should never be neglected where rheumatism is even suspected in a child. A confident opinion, too, should not be expressed as to the freedom of the heart until a period of six months, or even a year, has elapsed after a severe attack of rheumatism, as the evidences of endocarditis may be very insidious and long in developing. The earliest indication of endocarditis is usually a blurring or prolongation of the first sound at the apex, and an accentuation of the second sound at the base. The blurring of the first sound frequently clears up under strict rest in bed, but in other cases is replaced by a definite systolic murmur. In heart cases of some standing, and usually where there has been more than one attack of rheumatism, it is not uncommon to find the systolic bruit at the apex preceded by a præsystolic and diastolic one. It is very uncommon, however, to find the long "purring" præsystolic murmur of adults in children under the age of puberty, this, perhaps, being due to the fact that the heart condition associated with it takes several years to develop.

Pericarditis.—This is less frequent than endocarditis, and indeed is usually accompanied by the latter. Pericarditis may, however, occur with positively no present or future evidence forthcoming of endocarditis. It usually occurs with the more definite signs of rheumatism, such as arthritis, and subcutaneous nodules are present in a large proportion of cases. In rare instances pericarditis is found where the other evidences of rheumatism have been so slight and evanescent as to escape notice. Such cases are extremely exceptional, and the number of them has been exaggerated by some writers, who have also strained the analogy between the pericardium and a joint in attempted explanations of them. The pyrexia is generally greater with pericarditis than with any other phase of rheumatism in children, and the temperature frequently ranges from 104° to 106°, or even higher. Pericarditis is usually present, too, in the unusual occurrence of death ensuing directly from the rheumatic attack. In all cases of pyrexia in children, with no direct obvious cause, the suspicion of pericarditis should be entertained, as the symptoms of pericarditis may precede or obscure all other manifestations of rheumatism. Rheumatic pericarditis very seldom leads to any great effusion into the sac. Cases of apparently great distension of the pericardium with effusion are generally ones of thickened membrane with dilated and hyper-

trophied hearts, and the criteria between the two conditions, such as the position of the apex beat and dulness to the right of the sternum in the fourth rib interspace, are unreliable when applied to actual practice.

Affections of the Cardiac Muscle.—In endocarditis and pericarditis the inflammation may spread to the contiguous layers of the cardiac substance by direct extension. In the slighter degrees of inflammation of the cardiac muscle resulting in this way, there are probably no physical signs traceable to it beyond those of the endocarditis or pericarditis responsible for it. Lees and Poynton have, however, shown by post-mortem evidence that a general and intense myositis may arise in the early and acute stages of rheumatism. Sturges, too, believed that a rheumatic carditis was a frequent occurrence in children, in cases where only the usual signs of endocarditis or pericarditis were present. A grave affection of the cardiac muscle should be suspected in the active stage of rheumatism when there is marked dyspnœa, a diffused and feeble apex beat, along with a frequent, feeble, and intermittent pulse, and there is no well-established valvular disease to otherwise account for such symptoms. In an exceedingly able article (Allchin's *Manual of Medicine*, vol. i.) Lees states that acute dilatation of the left ventricle is almost invariable in the rheumatism of children, the more marked cases being due to a definite myositis, and the slighter degrees of dilatation to a toxic action on the cardiac muscle. This acute dilatation, he insists, is in no sense a "complication"; it is a characteristic, and the most important part of the disease. But it may be said that the evidence he brings in support of this important proposition, increase in size of the deep cardiac dulness, is hardly convincing, as the limits of the deep cardiac dulness would probably vary in any one case with the powers of percussion of different observers, and even with those of the same observer from time to time. The changes in the cardiac muscle, found along with hypertrophy and dilatation, in cases of long-established valvular disease, have a direct origin in the valvular defects, and by no means imply any pre-existing inflammatory state of the muscle.

Chorea.—That there is a close association of chorea with rheumatism is not to be doubted. The exact relationship between the two complaints is still a vexed question, but the more it is looked into the more intimate the association is found to be. With a large number, indeed, of competent observers, chorea is regarded in every instance as simply and solely a manifestation of rheumatism. Not infrequently choreic symptoms, of slight or greater intensity, are found in children suffering from rheumatism. Very often, too, chorea will attack children shortly after the subsidence of a definite rheumatic attack. Again, children who have suffered from chorea, with no present or past evidences of rheumatism, are much more prone to future rheumatic attacks than others. It is not uncommon, moreover, to find endocarditis in choreic children with no history of joint pains or other rheumatic symptoms; and, but much more rarely, cases occur where chorea is associated with endocarditis and rheumatic nodules where other evidence of rheumatism is entirely wanting. These facts are incontrovertible and fully admitted, but they are totally insufficient to support the dogma that in every instance chorea is a direct manifestation of rheumatism. In the great majority of cases of chorea no evidence of rheumatism, either in the past or present, is obtainable. It is very common, too, for children to have repeated and frequent attacks of chorea, and in whom no endocarditis or other symptoms of rheumatism develop. The proportion of cases of chorea associated with rheumatism has been variously stated by different authorities—the variance in the figures being doubtlessly influenced by the fact of the observer being a believer or disbeliever in the pan-rheumatic origin of chorea. Thus Dr. Still, in 226 cases of chorea, found evidence of rheumatism in at least 55·7 per cent. Other good observers, with a full knowledge of how vague and fleeting the symptoms of rheumatism in the child may be, feel justified in placing the percentage of cases of chorea associated with rheumatism at as low a figure as 30 per cent. These last figures will have to be largely discounted, of course, by the fact that possibly many of the cases of chorea contracted rheumatism after passing out of observation, and that many of them, too, were possibly seen before the age at which some of the later evidences of endocarditis, such as mitral stenosis, had had time to develop. After making very liberal allowance for these considerations, however, it probably remains true that more than half the cases of chorea pass through life without exhibiting any of the more definite signs of rheumatism. If, then, chorea is merely a phase of rheumatism, in the majority of cases it stands alone in this last respect. In a disease like chorea, where the beginning and termination are often so ill-defined, and where there is so wide a variance in the severity and duration of the complaint, it is difficult to attempt any discrimination between one group of cases and another. On the whole, perhaps it may be found that in the cases associated with definite rheumatism the onset is better marked, the acme as regards extent and violence of the movements sooner reached, and the duration shorter than in the cases where there is no evidence of rheumatism obtainable. The explanation of the frequent association of the two complaints is perhaps to be found in the neurotic predisposition, which even the most

ardent advocates of the infective origin of rheumatism are forced to admit.

Diagnosis.—This is generally not difficult where arthritis is present, and rheumatism is by far the most frequent cause of arthritis in children. The subsidence of inflammation in one joint with its reappearance in another is extremely characteristic of rheumatism. The multiple arthritis secondary to exanthematous disease may lead to error if no reliable history be obtainable. But in such cases the arthritis is seldom so marked as it is in rheumatism when several joints are simultaneously affected. The symmetrical arthritis occurring in the subjects of inherited syphilis should not mislead, as the joint affection is too chronic and too little painful to own rheumatism as a probable cause, and in a large percentage of such cases, moreover, Hutchinsonian teeth and interstitial keratitis are present. Gonorrhœal arthritis is rare, but by no means unknown in children, and, in the absence of the characteristic discharge, it is only the persistence of the joint affection in spite of the administration of salicylates that may negative the diagnosis of rheumatism. In the absence of evidence of a source of infection, pyæmic arthritis may present great difficulties in the way of discrimination from rheumatism. The greater local inflammation and reddening of the skin, the longer persistence, and the nature of the temperature generally, eventually lead to the correct diagnosis. Acute arthritis sometimes occurs with the onset of anterior poliomyelitis. The age of the child, the restriction of the swelling usually to one joint, the paralysis of the affected limb, and the rapid onset of muscular wasting are generally sufficient to avert mistakes. Infantile scurvy is sometimes mistaken for rheumatism, but the error should not occur where the age of the child, the other evidences of scurvy, and the fact that the swelling is primarily, at least, not in the joint, but over the shafts of the long bones, are taken into account. The swelling of the joints sometimes arising in association with syphilitic epiphysitis is hardly likely to be confounded with rheumatism if any care be exercised. Rheumatoid arthritis, a rare disease in children, may be mistaken for rheumatism in early acute stages. The resistance of the arthritis to the salicylates, the persistence of the complaint to certain joints and the freedom of others, the early muscular wasting, and the characteristic deformity about the joints seldom permit the error to be one of long duration. Apart from arthritis there is hardly anything characteristic about any of the other symptoms of rheumatism, and their nature is generally only settled by a history of arthritis in the past, or its subsequent appearance, or their association with fibrous nodules or endocarditis. It may be said, however, that growing pains of sufficient severity to cause limping are very seldom due to any other cause than rheumatism.

Prognosis.—Death is somewhat uncommon during the acute stages of the complaint, and exceedingly rare in a first attack. In attacks other than the first, the prognosis should be founded on the extent any persisting heart affection has attained, rather than on any special feature of the active complaint. Pericarditis, however, is attended with especial dangers of its own, and is nearly always found in those dying during an acute attack. A combination of severe chorea with active rheumatism is of bad augury, as in such cases the temperature is apt to run up high, and recovery seldom ensues. Numerous or persistent nodules add much to the gravity of the attack. Large nodules usually foretell death at no distant date, and Cheadle is right in the main when he states that the sight of them is the equivalent of that of the death warrant of the patient. Apart from these considerations, recovery is the rule whether the heart be left permanently damaged or not. If the prognosis as regards recovery from acute attacks be generally good, that as regards the recurrence of future attacks is correspondingly bad. Once rheumatism, always rheumatism, is not true, for even a young child may have a solitary attack of rheumatism and no more. But such cases are the exception, and usually a first rheumatic attack is usually the prelude to others in the near or distant future. Every succeeding attack adds to the gravity as regards the heart, either by originating endocarditis, or by adding its own quotum to a damage already sustained.

Treatment.—When any definite active rheumatism is present the child should be strictly confined to bed. He should be clothed in flannel, and rest between blankets instead of the ordinary linen sheets. Salicylate of soda should be given in doses of about three-quarters of a grain for each year of his age. This should at first be given every three hours in the day, and at night if awake, until the temperature returns to normal and all pains have disappeared. After this it should be continued three times a day for another fortnight at least. Should any toxic symptoms arise, such as delirium, vomiting, epistaxis, intestinal hæmorrhage, or albuminuria, then the quantity of the dose should be diminished, and the drug should only be entirely withheld in the case of absolute necessity. There is some advantage, probably, in giving some alkali, such as the bicarbonate of potash, along with the salicylate, in doses of two or three grains of the former to each grain of the latter. Painful joints should be swathed in flannel bandages, or better still in cotton-wool covered with oil-silk. Any obstinate arthritis is generally speedily benefited by small blisters in the neighbourhood of the joints, but young children are bad sub-

jects, as a rule, for blistering. If there is much pain, then a single full dose of opium, appropriate to the age of the child, given at bed-time, is often extremely beneficial in procuring rest and sleep. After the salicylates have been stopped, the child should be put on a course of iron, such as four or five grain doses of the double citrate of iron and ammonia, whether marked anæmia be present or not. To this a corresponding quantity of citrate of potash is often a useful addition. As far as possible a supine position should be maintained, and, if there be any suspicion of endocarditis, rigidly enforced. A change from the erect to the supine position means, on an average, ten beats less per minute, with a corresponding diminution in the labour of the heart. Not only this, moreover, but when endocarditis is present it means ten less flappings together of the inflamed valvular cusps, and a corresponding increase in the length of the diastole, the only time in the cardiac cycle when repair to its delicate structures is possible or probable. The course of salicylate should not be stopped on the advent of endocarditis. Any irregularity of pulse, or feeble cardiac action, is much more likely due to the disease than to the depressing effects of the drug, which have been much exaggerated. Such conditions are better met by the administration of strychnine, preferably by injection, rather than by the stoppage of the salicylate. Dr. Caton's statistics show such satisfactory results that his treatment deserves a trial in the early stages of endocarditis. This consists in giving appropriate doses of sodium iodide, and the repeated application of small blisters in the lines between the middle of the clavicles and the nipples on either side. Here again at first the salicylate should be given along with the iodide, as the baneful results of a relapse of the rheumatism are much more certain than that any benefit will be gained from the rest of the special treatment. In the early stages of pericarditis two or three leeches should be applied over the heart, or recourse may be had to the ice-bag so warmly advocated by Lees. Blisters, too, are probably useful, but their use interferes with a complete and satisfactory daily examination of the heart. When there is pain or distress no drug is so useful as opium, and this can also be given at first with the salicylate. Alcohol is very seldom required in any of the active phases of rheumatism in children. A child should be kept in bed for at least a month after the subsidence of an attack of marked rheumatism. This, however, is generally difficult of attainment, as the child, perhaps feeling quite well, resents the confinement to bed as irksome, and only too often the parents totally fail to appreciate the gravity of the complaint when once the severe symptoms have disappeared.

The child who has once had rheumatism should be clothed in flannel or woollen next to the skin, and the clothing is of more importance in the summer even than in the winter time. He should be restricted from the more violent games, such as running or football, not only because the fatigue they cause predisposes to rheumatism, but also because the fact of having had rheumatism should render his heart an object of suspicion, although he may present no evidence of valvular defect. If possible, he should not reside in low, damp places, such as the Fen country in England, where there is no doubt rheumatism is unduly prevalent. Apart from such notoriously unsuitable places, however, care and protection from fatigue and damp are more important than place of residence. Numerous places bear a reputation as being "anti-rheumatic" on evidence that will not bear investigation; whilst residence by the sea seems beneficial to some children, to others it is apparently detrimental. In fact, when the choice is restricted to these islands, a suitable place of residence is more or less a matter of chance. The prevalent belief that too much butcher meat is harmful for rheumatic children rests on some slight foundation, and it may be well to put the child on a diet where a slight deficiency in it is replaced by an extra quantity of milk, fish, vegetables, or other food. The practice, too, of giving the juice of a fresh lemon daily to a rheumatic child, in the shape of lemonade, can seldom do any harm, and it may possibly do good by ensuring the continuance of a mild alkaline course in a pleasant and acceptable form.

Rheumatoid Arthritis

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SYNONYMS.—Arthritis deformans, Polyarthritis chronica, Osteo-arthritis, Rheumatic gout, Mechanical or Traumatic arthritis, Arthritis chronica villosa.

Few diseases so common as rheumatoid arthritis, and of equal importance, are regarded by the profession with so little unanimity. This form of joint affection is known to be of great antiquity; the Museum of the Royal College of Surgeons possesses bones showing characteristic changes which date back to 1300 B.C. It has received the attention of genera-

tions of able workers, but even now wide diversity of opinion exists among observers of repute as to its pathology and true place in medicine. It seems possible that conditions widely different in origin, in clinical manifestations, and in results have been grouped together as rheumatoid arthritis, leading to the confusion apparent in writings on the subject.

An attempt will be made in these pages to describe the disease as it occurs in adults, and in certain varieties which are marked off from each other with some clearness. It is perhaps doubtful whether these groups will ultimately be placed together under one name, but for the present, tradition and convenience require that they should be considered side by side.

Cases presenting themselves for observation in adults fall into the following main clinical groups:—

1. Polyarticular rheumatoid arthritis: (*a*) Subacute and chronic; (*b*) Acute.
2. Localised or senile rheumatoid arthritis.

1. Polyarticular Rheumatoid Arthritis

(*a*) *Subacute and Chronic*

Etiology.—Age.—This form occurs at any age from childhood to late adult life. The age most prone to attack appears to be that between thirty-five and fifty, the period of commencing tissue degeneration. Cases occurring much earlier are common, and women are frequently attacked during the time of greatest reproductive activity. The records of the New Hospital for Women furnish repeated examples of this fact. A certain number of cases appear about puberty. On the whole, the earlier the time of attack the more severe and rapidly progressive is the course of the illness, but there are exceptions to this general truth. Some of the early cases improve strikingly under treatment, and the disease may even become arrested if it has been recognised early and dealt with at once.

Hereditary influence does not appear to be strongly shown in the history of this disease. Garrod considers that heredity has an unimportant bearing on its production, and is decidedly less marked in this affection than in gout; it is probably not to be traced with any frequency even in the rheumatoid arthritis of children. It is not very rare to find several female members of a family suffering more or less severely from rheumatoid arthritis, and a history can sometimes be obtained, especially in private practice, of a joint affection which sounds like rheumatoid arthritis in relatives in a previous generation. It is not unknown for several male members of one family to suffer from early and severe forms, but this is rare. Bannatyne analyses 293 cases, and finds a history of clear inheritance in 15 only. Statistics as to heredity, compiled from the statements of patients respecting the joint affections of their relatives, are open to fallacies so obvious as to greatly diminish their value.

As regards *sex*, there is undoubtedly a great and special liability among women to suffer. Badt gives a recent analysis of 53 cases observed by him. Of these 6 per cent were males, 94 per cent were females. In 80 per cent of the female cases the illness began at the climacteric. The influence of pelvic disease and of the menopause has been dwelt upon by many writers, and statistics show that these conditions are of great importance. At the climacteric the general health of women is lowered, and they are then liable to suffer from diseases of the reproductive system. Of such diseases probably those most potent in the production of rheumatoid arthritis are such as are associated with exhausting losses from the uterus, especially hæmorrhages from any cause, and, to a less degree, copious purulent or muco-purulent discharges. At the New Hospital for Women the occurrence of rheumatoid arthritis, more or less severe, in association with menorrhagia due to fibroid tumours of the uterus has been repeatedly observed. Such an association has been noted also by Ord. Other less definite conditions, such as uterine displacements or ovarian neuralgia, have been thought to bear some causative relation to the disease, but it is difficult to trace the evidence on which this opinion is based. Chronic ovarian pain seems a possible influence, and in one case had been a source of great suffering in an unmarried woman of thirty-two, who developed severe rheumatoid arthritis.

It has been suggested that the special liability of women at the climacteric may be due to a failure on the part of the ovary at that time to exercise its normal influence on general nutrition, and Badt suggests the use of ovarian extract. The idea belongs at present to the region of hypothesis, but deserves further investigation. There does not appear to be a special danger of the onset of the disease after the removal of the ovaries surgically. This point has been under observation at the New Hospital for Women, and so far the evidence is negative.

Garrod pointed out that rheumatoid arthritis occurs early and in severe forms in association with rapid child-bearing and over-lactation, and other observers have amply confirmed this statement. It is probable that any cause whatever which tends to lower the general health and to produce marked debility and failure of nutrition may predispose to the disease. In women diseases of the pelvic organs form so considerable a proportion of the causes of ill-health, that it is to be expected that these diseases should take a prominent place in the history of an illness liable to attack the debilitated from any cause.

Prolonged mental strain, long anxiety, and worry are very frequently present in the history of these cases, in association with other debilitating conditions. In some cases the history of mental worry or anxiety seems the most prominent fact in the history, and stands out alone, or as the chief factor which can be made out.

As a rule, the depressing mental conditions have been of long standing, going on for months or even years, but there is some evidence that a severe shock may be followed quickly by joint changes. The writer has notes of a case in a woman of thirty-five, in whom rheumatoid arthritis in a severe form followed immediately on the shock occasioned by the sudden death of her twin children. Cases of a like nature have been recorded by others.

Climate probably has some influence on the production of this complaint, as it undoubtedly has upon its course. Charcot considered damp cold to be one of the most potent factors. Bannatyne states that the disease is more common in the humid air of Ireland than in England; Adams that it is prevalent in Holland; and Stewart that it is very rare in the dry climate of Canada. In a certain number of cases the disease is referred to a definite exposure to cold or damp, or both, such as sleeping in a damp bed, or getting wet and sitting in damp clothes. The combination of damp, cold, a low elevation, and low average of bright sunshine appears to be favourable to its development—in other words, the climate least likely to lead to good general health and a high vitality of the tissues.

The influence of *traumatic injury* to joints has been variously estimated by writers. It is generally accepted as true that in the monarticular form previous injury may determine the localisation of the affection, even when the injury has occurred long before the onset of the joint changes. It is also true that in cases of moderate generalised rheumatoid arthritis an injury may greatly increase the activity of the disease in the injured joint, which may quickly become completely crippled, although the activity of the joint affection generally is not increased.

Cases are recorded where injury to one joint was apparently followed by generalised rheumatoid arthritis. The evidence in those cases available for reference is not absolutely convincing, and in some the length of time which had elapsed between the injury and the onset of the generalised disease was so great that the influence of the injury appears somewhat doubtful.

There is some evidence that joints which have previously been attacked by the inflammatory processes of acute rheumatism are prone to be affected later by rheumatoid arthritis. Luff states that acute rheumatism is an undoubted predisposing cause of this disease. Modern views as to the pathology of these affections tend to minimise rather than to accentuate the points of difference between them. If acute rheumatism, and certain forms at least of rheumatoid arthritis, have a bacterial origin, as some believe, it is possible that the tissues of a joint already damaged by one such infection may readily become a nidus in which other organisms can flourish. It is certain that the changes characteristic of chronic rheumatoid arthritis may supervene in other chronically inflamed joints, *i.e.* those of gout or of gonorrhœa.

A low *diet*, deficient in nutritive elements, may be an important element in the causation of rheumatoid arthritis. Alcohol has not the same directly deleterious effect on these patients as on the sufferers from gout.

Clinical Characters of Subacute and Chronic Polyarticular Rheumatoid Arthritis.—The onset is insidious and difficult to date from the accounts given by patients.

Premonitory symptoms have been described by some writers, such as abnormal sensations in the extremities, tingling, pins and needles, and numbness, especially on waking from sleep. These sensations are frequently complained of early, but examination shows that some joints are already affected, and pain and enlargement of joints are the earliest distinctive symptoms. The joints most liable to attack are those of the hands, and, broadly speaking, the disease assumes a symmetrical arrangement, attacking corresponding joints on the two sides, and tending to advance from the extremities towards the trunk. The number of joints affected varies greatly, and there appears to be a process of selection which may be different in different cases, but is usually the same on the two sides of the body. The joints commonly attacked are the interphalangeal joints of the hands, or some of them, the metacarpo-phalangeal joints, the wrist, elbow, and shoulder. In the lower extremities the knee is very frequently attacked, also the ankle, the small joints of the foot, and the hip. The temporo-maxillary joint is frequently affected—a fact which is valuable in diagnosis, and serves to distinguish this from other forms of arthritis, except that which is the result of gonorrhœal infection. Involvement of the joints of the vertebræ occurs, especially of the occipito-atloid joint, resulting in stiffness or definite limitation of nodding or rotatory movements of the head. The pain due to involvement of the spine may be very severe.

Although marked symmetry of arrangement is the rule, and is a characteristic feature of the disease, it is by no means invariable. The joints on one side of the body may show evidence of advanced changes, while those on the other side are only slightly affected, and there are also exceptions to the rule that the disease tends

to advance in order from periphery to trunk. Joints may be picked out for attack without reference to anatomical sequence (Fig. 1). Sym-



Fig. 1.

metry is more apparent in the earlier and more rapidly progressive cases. In those less active the disease shows a tendency to remain localised in a few joints.

The changes, once begun, have no spontaneous tendency to recover, and the joint becomes the seat of a gradually progressive disorganisation, unless the disease is arrested by treatment.

The actual condition of the joint varies. Clinically, two main varieties of joint change are noticeable.

(*a*) The joint assumes the shape of a more or less symmetrical truncated spindle, it is swelled, its normal contour is obscured, and its bony landmarks are difficult to make out. The impression received on examination is that the soft parts, both within and without the joints, are uniformly swelled and thickened. This form is well seen in the finger-joints, and is shown in Fig. 2. The joint is tender to touch,



Fig. 2.

sometimes even hot, and movement is painful and limited. The terminal inter-phalangeal joints frequently escape attack. Not rarely the joint contains fluid, and definite fluctuation is present. In other cases no fluid can be made out, but the joint has a characteristic doughy, semi-elastic feel. The metacarpo-phalangeal joints suffer frequently, especially those of the first and second fingers. That of the little finger is usually less affected. No bony out-growths may be present, no lipping, nor any gross bony changes. The condition is suggestive of a chronic inflammatory over-growth of the soft parts of the joint, especially of the synovial membrane. Schüller proposes calling this form chronic villous arthritis (arthritis chronica villosa).

(*b*) The other type of joint change presents a striking contrast to that described above. The joint is enlarged, but the enlargement is irregular and often nodular. The bony landmarks are more, rather than less, prominent, and there is obvious bony enlargement; lipping of the articular surface, and definite osseous out-growths can often be made out. Tenderness on manipulation is not usually very marked, though some areas of the joint may be decidedly tender on deep pressure. Movement is limited and painful, and may be attended with marked grating, due either to actual contact of bony surfaces, or more often, probably, to creaking of thickened periarticular tissues. The joint may become completely immobile and apparently ankylosed, or the bony out-growths may be so situated as mechanically to lock the joint. There is usually no excess of fluid in these joints. This form also can be well studied in the hand, especially in the metacarpo-phalangeal or carpo-metacarpal joints of the thumb. The terminal phalanges may show Heberden's nodes at their bases. This type is shown in Fig. 3.



Fig. 3.

Speaking generally, the first group represents the more acute form of the disease and occurs in younger patients, the second the more chronic form.

In some late cases the joint shows a combination of bony enlargement or nodosities, with increased thickening of soft parts and fluid effusion, but it is certain that joints may exhibit typically the bony changes last described without having passed through a stage of general spindle-like swelling or fluid effusion. The two varieties have clinically well-defined characters which merit separate description, and it seems certain that spindle-shaped swelling is not merely the early stage of an arthritis which passes inevitably into a condition of bony out-growth and enlargement.

(*c*) In a third condition occasionally seen, some joints of the fingers, especially the terminal inter-phalangeal joints, seem actually smaller than usual, and their movements more than normally free. They are painless, and

look as if there had been some atrophy of all the parts concerned—bones and soft parts alike. This condition is present in association with spindle-shaped enlargement of joints rather than in the more chronic type with bony outgrowth.

The anatomical changes in the joints are invariably associated with more or less *atrophy of muscles* of the limb; sometimes the atrophy is extreme, and adds greatly to the general deformity. The extensors of the joints suffer usually more than the flexors. In the hand the interossei muscles suffer, especially the dorsal interossei. The thenar and hypothenar eminences do not escape, and the general wasting of muscles of the limb may be so decided as to simulate the changes in progressive muscular atrophy.

The reaction of degeneration is usually absent. The wasting of muscles bears no necessary proportion to the degree of involvement of the joint, and it may be very marked where the joint changes are slight or moderate. Fig. 4 shows a case with extreme wasting of the muscles of the leg. The tendon reflexes in the affected limbs are frequently, but not invariably, increased.



Fig. 4.

The *deformities* resulting from the joint and muscle changes have been studied and described by many writers. In the hands a common and early change is ulnar deflection of the fingers at the metacarpo-phalangeal joints. At first the deflection is slight and easily reduced, later it may become extreme, and only partially or not at all reducible. The other deformities of the hand depend on flexion and extension of the phalanges on each other, or on the metacarpal bones. The knee joint in advanced cases is almost invariably flexed. The ankle joint is frequently fixed, the foot abducted, and the great toe deflected to the outer side. The temporo-maxillary joint may be ankylosed, and the patient unable to masticate; and the head may be fixed, either in a natural position or in a position of flexion or rotation, from changes in the cervical spine.

The causes of the deformities are briefly either—

(*a*) Ankylosis or partial ankylosis of joints, or changes in the form of the articular surfaces.

(*b*) Atrophy of certain groups of muscles and increased tonicity or spasm of others, leading to disturbance of normal relations of muscular groups to one another, and in extreme cases causing actual dislocation of bones, partial or complete.

(*c*) Changes in the ligaments and tendons about the joint, commonly in the direction of increased laxity or destruction.

(*d*) The effect of gravity and habitual posture.

Most of the deformities of rheumatoid arthritis are not peculiar to it, but may be present also in other chronic affections of the joints, such as gout, and in various diseases of the central nervous system where the nutrition of muscles and joints is affected.

Polyarticular rheumatoid arthritis does not consist only in the joint changes and their results, but presents other clinical features of interest.

The *temperature* is usually normal, but even in the less acute cases may show irregular evening rises of one or two degrees, with occasional more decided fever. Such pyrexial periods may alternate with times when no rise is noted, and do not appear to be connected necessarily with obvious extension of the disease.

The *pulse* varies. It has been pointed out by Spender, Dyce Duckworth, and others, that a rapid pulse may be present, and the former author attaches great importance to the fact, and considers it to be of very frequent occurrence and of diagnostic value. He speaks of a pulse-rate of 110, 115, 120, and even 140 in apyrexial cases, and in association with coldness of the surface.

The opinion of clinical observers generally will probably be that such decided persistent rapidity of the pulse as this is not the rule, and it is an undoubted fact that, even in cases showing slight evening fever, the pulse-rate may not rise above 90 for weeks together. In some cases there is irregularity of the pulse, and it tends to become temporarily quickened by slight causes.

Anæmia is present in most cases to a greater or less extent. It is usually moderate in degree, persistent, and not easily amenable to treatment. In some cases the anæmia is very striking. The hæmoglobin value of the individual red corpuscle is reduced, and there may be some relative increase of white cells. The presence of anæmia is considered by some writers as evidence of bacterial toxæmia. Bouchard gives 91 cases of anæmia in 1000, a proportion smaller than experience in this country would suggest.

The *lymphatic glands*, especially those of the axilla and groin, may be enlarged in certain cases in adults. Bannatyne states that he has found a chain running up from affected joints. He has never found an enlarged spleen. Still and others mention enlargement of glands in children, especially those of the axilla and groin. In children the spleen is enlarged in one, at least, of the conditions called rheumatoid arthritis.

The state of the patient as regards *pain* varies

greatly in different cases, and at different times in the same case. Pain is often severe and persistent, worse at night, and aggravated by movement and by changes in the weather. In other cases the patient complains chiefly of great stiffness of joints, especially after sleep or resting. Rarely true neuralgia of a severe type is present, and in those cases the bouts of pain have, in the writer's experience, tended to occur in the night after a first sleep. In Bouchard's analysis of 1000 cases neuralgia occurred in 30.

Stiffness of joints may be an early sign, and frequently appears before pain is complained of. It should always suggest the possibility of rheumatoid arthritis.

Evidence of *trophic* or *vaso-motor* changes of the skin is common. The skin of the hands and feet may be cold, blue, and clammy, and a combination of cold hands and feet, with profuse sweating of the palms and soles, is striking and characteristic. Sweating, either general or confined to some one area, such as the hands, feet, upper lip, etc., is common, and patients sometimes complain of exhausting nocturnal sweats. The skin may show areas of definite atrophy, especially near affected joints, such as "glossy skin." There may be patches of congestion and distressing sensations of burning or pricking. Spender and others have laid stress on pigmentation of the skin. It is present in a large number of cases, but whether there is any form of pigmentation of the skin peculiar to rheumatoid arthritis, either in tint or distribution, is perhaps doubtful. Pigmentation occurs in connection with many chronic diseases, especially those associated with failure of nutrition, but also in apparently healthy subjects. It occurs under such varying pathological conditions that it can hardly be considered as diagnostic, but should, perhaps, be regarded as an incident in numerous conditions of ill-health, of which rheumatoid arthritis is a notable example. Pigmentation occurs as—

(*a*) A general darkening of the complexion, most marked usually on exposed parts.

(*b*) As large patches of pigmentation in various parts, of irregular shape, uniform in colour, and with well-defined edges.

(*c*) As more or less widely spread small patches with a general appearance like freckling, or splashes of paint, not usually most marked on exposed parts, but on the trunk and limbs, especially, sometimes, on parts exposed to pressure, such as the waist, garter areas, etc. In this form there may be decided symmetry in the arrangement of the patches. The tint varies from pale honey shades to dark brown or black, and is influenced to some extent by the natural complexion of the patient.

Bannatyne mentions the occurrence of subcutaneous hæmorrhagic effusion, and also under the nails.

The nails are often dull, brittle, and show longitudinal striations.

Œdema is occasionally present either in patches near joints or of dependent parts.

Neuritis may occur leading to severe pain along a nerve trunk, and followed by rapid wasting of the muscles supplied by it, with loss of faradic excitability. When, in this disease, loss of faradic excitability is present, it is usually restricted to one or more muscles, and is probably the result of secondary neuritis.

Garrod and others have thought *psoriasis* was apt to occur with more than average frequency in association with rheumatoid rheumatism.

In children slight *exophthalmos* has been noted.

Bouchard finds *dilatation of the stomach* in no fewer than 273 cases in 1000.

The condition of the *heart* is a subject about which a good deal of diversity of opinion exists. Recent work has revived interest in this point, but a definite conclusion is difficult to arrive at.

Garrod considered the absence of cardiac inflammation as one of the most important diagnostic features of rheumatoid arthritis, and said he had never traced the occurrence of pericarditis or endocarditis in this disease. Charcot believes that both may occur, and brings post-mortem evidence to prove the existence of endocarditis. In one, at least, of the diseases called rheumatoid arthritis in children there appears to be a remarkable tendency to inflammation of the pericardium, and post-mortem examination in these cases shows adherent pericardium with striking frequency. In the account of a post-mortem examination on a patient aged thirty-seven recently published, it is stated that the surfaces of the pericardium were adherent.

In acute cases there may be clinical evidence of endocarditis or pericarditis. Bannatyne publishes two cases (*Brit. Med. Jour.* 1898) in which these complications were a marked feature. In one case observed by the writer there was pericardial effusion. In the larger number of cases no clinical signs of heart complications are present, but in some there are well-marked cardiac murmurs. These have hitherto been ascribed to the results of previous attacks of true rheumatism. In some cases, no doubt, this explanation is the correct one, in others no evidence supports it. It is possible that when the significance of acute attacks is more generally appreciated, an illness which formerly would have been ascribed to acute rheumatism will be regarded as acute rheumatoid arthritis.

It is worthy of note that in enumerating the diseases associated with "chronic articular rheumatism," in 1000 cases Bouchard records no fewer than 60 cases of aortic disease, and 91 of hypertrophy of the heart. These figures

suggest the need of further observation of heart conditions in this disease.

The *urine* usually presents no important changes from the normal. Angel Money found excess of urea and uric acid, and occasionally glycosuria. Drackmann and Böcker both found diminution of phosphates. Schüller constantly finds a diminution of calcium salts and earthy phosphates, a low specific gravity, and a reaction either faintly acid or neutral in the variety he calls arthritis deformans, *i.e.* where bones rather than soft parts are chiefly affected. Van Noorder, Rumpff, and others have noted similar changes, and Schüller considers them characteristic. Bain (*Edin. Med. Journal*, 1900) analysed the urine in one case only, and found diminished excretion of uric acid.

Occasional accompaniments of rheumatoid arthritis, which have received a good deal of study, and which frequently occur in the same subject, are—

(*a*) A chronic bursitis with thickening of the walls of the bursa, and some fluid effusion. This is well seen in the bursa over the olecranon, and may be present near joints in both the upper and lower extremities.

(*b*) Subcutaneous fibrous nodules, resembling in some respects the nodules which occur in true rheumatism. Fig. 5 shows both these conditions in a marked degree. The bursa over the olecranon on both sides formed a visible tumour, and its walls were greatly thickened. Several hard bodies could be felt within its cavity. The swellings were painless, and had always been so.



FIG. 5.

The smaller swellings were due to subcutaneous fibrous nodules, oval, flattened, painless, and freely movable.

These nodules differ from those characteristic of true rheumatism in certain particulars.

1. They appear in late life.

2. They persist longer than is usual with those of acute rheumatism. In the patient shown in Fig. 5 the nodules are known to have remained unaltered for three years.

3. The history of acute rheumatism is not usually obtained, nor is there any evidence pointing to its presence, or to the presence of active and progressive cardiac complications.

While in hospital the patient referred to above developed a new nodule on the right olecranon. It was at first the size of a hemp seed, decidedly tender on pressure, and grew very slowly. It felt precisely like a true rheumatic nodule. In this case the nodules were confined to the arms. There was no history of acute rheumatism.

Ganglion-like distention of the sheaths of the tendons of the muscles of the hands have been described.

The *course* varies within very wide limits. The disease may progress with appalling rapidity, one joint after another being quickly involved, until the patient becomes a helpless cripple, unable to walk, stand, or feed himself, and with many joints fixed and useless. Such severe cases occur usually in younger patients.

The affection may be much less acute than this, and for years after the nature of the illness is recognisable the patient is able to live an ordinary life, complaining only of pain, stiffness, and some disfigurement of joints. The activity of the affection varies at different times, and conditions affecting the general health adversely also make the joints worse.

In some cases in elderly subjects no complaint of pain is made, though the hands show to a typical degree the bony changes which have been described, and are markedly deformed. There is no evidence that such hands have ever passed through a stage when there was spindle-shaped swelling of the joints of the fingers or pain. The patients have usually laboured hard, and the hand presents the character which Lane has described as mechanical or traumatic arthritis. Washerwomen exhibit this condition typically.

(*b*) *Acute Polyarticular Rheumatoid Arthritis*

The occurrence of an acute form has long been known and its clinical characters described. Later work has emphasised the importance of this variety of the disease, and it has received careful study, especially as regards etiology, since the possible bacterial origin of rheumatoid arthritis has engaged the attention of authors.

Although no hard-and-fast line can be drawn between this form and the more acute cases of the so-called chronic variety, certain features are worthy of attention.

As regards etiology.

Age.—It occurs before middle life, chiefly in children and in young adults, but is not unknown later. The case shown in Fig. 1 began at thirty-nine.

It is a notable fact that some, at least, of the cases occur in definite association with septic infection. Suppurative tonsillitis, otitis media, suppuration in bone, behind the plate of false teeth, and gonorrhœa, have been present in the history of these cases. The influence of pyorrhœa alveolaris and other septic con-

ditions of the mouth in the production of disease has been insisted on by Hunter and others, and it is possible that oral sepsis may not be without importance in this connection. In one acute case in a girl of twenty recently under the writer's observation the gums and buccal mucous membrane were in a highly septic condition.

The *clinical characters* of the acute cases are as follows:—

The *onset* is sudden, or at least definite—the patient can give a precise account of the beginning of the illness.

There is decided fever, the temperature rising to 101°, 102°, or even higher each night. The affected joints are painful, tender, and swelled—there is effusion of fluid into them.

So far the resemblance to acute rheumatism is close, but the following points will help to distinguish this affection.

The joints affected are usually the smaller joints, and there is not the same tendency to migration which is characteristic of the arthritis of true rheumatism. The joints attacked do not return to the normal, but further changes develop in them, and the spindle-shaped swelling of the finger joints already described quickly appears. The following case is a characteristic example of this affection:—

Woman. Aged twenty-six. Married. One child.

Previous health good, but for six months before present illness the patient had repeated attacks of tonsillitis with "gatherings" on the tonsil. She was suddenly attacked with joint pains, the finger joints and wrists were affected, and later the shoulder and knee.

When admitted to hospital the proximal inter-phalangeal joints of the fingers were swelled, painful, and contained fluid. Temperature 101°; pulse 90.

There was a soft systolic bruit to be heard at the apex.

The patient was treated by salicylates with little benefit, and the hands quickly showed characteristic changes, such as are described above, and illustrated in Fig. 1.

She left the hospital after six weeks' stay with these changes distinctly present in the hands and wrists, with no evening temperature, and with the cardiac bruit still audible; pulse 80.

There was no history of a previous attack of acute rheumatism in this case.

2. Localised, Senile, or Monarticular Arthritis

In this variety of joint affection there are clinical points which mark it off in a striking way from the other varieties which have been described. So decided are the differences, indeed, that they are more evident than the points of likeness.

The disease is essentially a local affection, has little tendency to symmetrical arrangement, and none to become generalised. It attacks those in late middle life or in old age. There is no pyrexia or evidence of constitutional disturbance. It frequently follows upon injury, and is more common in men than in women. The larger joints near the trunk, the hip, and the shoulder especially, are most apt to suffer—a fact which contrasts sharply with the localisation of the generalised polyarticular form, in which the small peripheral joints, more particularly those of the hands, are attacked with a frequency so great as to constitute a diagnostic feature. The affection is usually localised in one joint, but occasionally the corresponding joint on the opposite side may be affected in a lesser degree.

When the hip is the joint affected there is pain, stiffness, and limitation of movement. The patient walks lame, and finds it difficult or impossible to cross one leg over the other. The amount of actual change in the joint detected by examination varies. In muscular or fat subjects the hand may detect little, but limitation of movements and possibly some deep-seated grating, or shortening and eversion of the limb may be made out. In thin subjects definite changes in the anatomical outline of the joint can usually be felt, with irregularities due to bony changes, the "condyloid crests" of Hutchinson.

In the neighbourhood of such joints, as also in connection with Charcot's disease, the cysts described by Morant Baker may be present.

In the case of the shoulder, where the joint is more within reach of examination, changes in the articular surfaces can often be readily made out.

It is not certain whether the enlargement of the normal prominences on both sides of the terminal phalanges of the fingers on their dorsal surface, called Heberden's nodes, should be described in this place. If they are to be included at all among the manifestations of rheumatoid arthritis, it seems suitable that they should rank with the most chronic forms included under this group. They occur alone, or in association with the monarticular or very chronic generalised forms, rather than with the more acute disease, and may be conspicuously absent when the other finger joints show decided spindle-shaped swelling. There is seldom much pain, but the joint may be stiff and the disfigurement considerable. Deflexion of the terminal phalanx to the radial side is a common deformity. Great difference of opinion has existed among authors as to the pathology of these little growths, which have been variously regarded as of a gouty nature, or as due to rheumatoid arthritis. They certainly occur in the chronic forms of rheumatoid arthritis, and have been found also where there is a gouty history.

Garrod states that he has never seen them in true gout, and they have little in common with any other known result of gout.

Still has twice seen similar nodosities in children with congenital syphilis.

It is noteworthy that Bouchard quotes the diseases associated with Heberden's nodes in 1000 cases, and rheumatoid arthritis does not occur in this list.

Much light is thrown upon the production of permanent changes in the joints of the elderly by the well-known writings of Arbuthnot Lane. He does not accept the view that the changes are due to disease, but believes them to be "produced mechanically and in obedience to simple general laws." It is difficult to trace to this cause the acute forms of rheumatoid arthritis—those associated with fever, with effusion into joints, and with the characteristic changes which are included in Schüller's definition of arthritis chronica villosa. It is possible that a further classification will be adopted, and a distinction as regards pathology will be drawn between such cases as are included in Lane's description and the more acute generalised forms. Changes similar to those described by Lane are found in the lower animals, notably in old horses which have laboured heavily. Whether the acute form occurs in the lower animals is doubtful, and Bannatyne suggests that the failure of inoculation experiments may be due to the fact that the animals subjected to experiment were immune, and that rheumatoid arthritis as an acute affection does not occur in them.

Morbid Anatomy.—The changes in the interior of a joint affected by rheumatoid arthritis are as follows:—

(*a*) The articular cartilage is affected early—some think it is the primary change—the surface loses its polish, becomes dull and velvety, owing to splitting of the matrix into fine vertical fibrillæ, and destruction and disappearance of cartilage cells. The change is most marked towards the centre of the cartilage, and may extend and deepen until in the central part its whole thickness is destroyed, and the head of the bone beneath laid bare. In addition to this disintegration a process of irregular formation takes place at the periphery of the articular cartilage, leading to the lipping or nodulation common in later stages. In these out-growths bone may afterwards be deposited and osteophytes formed. This over-growth is considered by Cornil and Ranvier to be due to the fact that the outer edge of the cartilage is overlapped by folds of synovial membrane, which prevent the escape of cartilage cells into the cavity of the joint. Probably, also, the mode in which pressure is transmitted through the joint determines the site of greatest destruction of cartilage—the cartilage being destroyed chiefly "at the point of greatest stress." In old-standing cases the articular cartilages may be absorbed, and even the inter-articular cartilages, in such joints as the knee, wrist, or jaw.

(*b*) The bone beneath shows a thin layer of greatly increased density; it is smooth, polished, and ivory-like (eburnated). Its surface may be more or less deeply furrowed by the steady friction of prominences on the opposite bone. The lipping and the position of osteophytes may more or less completely destroy the mobility of the joint, or alter its mechanism so as to render possible movements which could not be performed by the normal joint. The osteophytes themselves may be worn away or eburnated.

The joint may be fixed by true bony ankylosis, which occurs in the spinal column. In affected joints where the cartilage is destroyed the articular surfaces have been described as united by bridges of cancellous bone easily broken down by slight pressure. Fibrous ankylosis of joints is not uncommon, but true bony ankylosis in the joints of the limbs is not the rule. It is only likely to occur in bedridden patients whose joints are at rest and much disorganised.

If the head of the bone is sawn through it is seen to be of an unusually spongy consistence, and to contain a large proportion of oily matter, as if from a species of fatty degeneration.

The condition of the synovial membrane is variously described. In a joint showing the above changes both the synovial membrane and the fibrous capsule of the joint will be greatly thickened, its fringes hypertrophied, and many will be seen to have undergone fatty changes, or to contain cartilaginous or osseous masses. These may become detached and form loose bodies in the joint. The bursæ near joints may show marked thickening of their capsules, and form decided swellings, and may contain hard bodies in their cavities. This is common in the bursa over the olecranon (Fig. 4). Bursæ may entirely disappear. In two shoulder joints from the same subject lately examined by the writer the subacromial bursæ had entirely disappeared, and the head of the humerus articulated directly with the upper part of the glenoid fossa and with the acromion. The ligaments of the joint may become thickened or stretched and elongated. Tendons entering into the structure of the joint may be entirely or partially destroyed, such as the ligamentum teres of the hip, or the biceps tendon of the shoulder. The muscles about joints may find new attachments to capsule or ligaments rather than to bony points. This was the case in the shoulder joints already mentioned.[1]

Suppuration is of extreme rarity, though a few cases are on record, and in one remarkable case under the care of the writer a condition resembling pyæmia, with suppuration in many joints, supervened while the patient was under

[1] For the opportunity of examining these joints the writer is indebted to Miss Stoney, M.D.

observation in hospital. No cause capable of producing pyæmia was discovered. Effusion of blood into the joint is referred to by Goodhart.

The above description can hardly be considered to apply with any closeness to a large number of cases of acute onset and rapid course, which, clinically, do not present the features which are detailed above. Bony out-growth and deformity are absent in such joints, and their absence is evident not only to the fingers of the examiner, but also by the more convincing test supplied by the X-rays. A skiagram from such a case is described and figured by Bannatyne.

A description of the joint tissues removed by operation has been given in detail chiefly by Continental writers, and it throws an important light on certain features of the disease. Schüller, of Berlin, has described the appearances in joints operated on by himself. He proposes to call a group of cases which he believes to have distinctive features "Arthritis chronica villosa," and reserves for chronic cases with changes in the bone and cartilage the name Arthritis deformans.

The essential feature in the former is chronic inflammatory over-growth of the synovial membrane, with the formation of fringes and tufts much larger and more massive than in normal joints, or in those the seat of other inflammatory processes.

In freshly opened joints the synovial membrane shows red, swollen, and highly vascular. He has never found in these cases the changes of the cartilage characteristic of arthritis deformans. In this form, and in this form only, he found a peculiar bacillus in the villi, to be described later. It is interesting to remember in this relation Bannatyne's statement in 1898, that his bacillus was found only in the acute polyarticular cases with soft synovial enlargement, and not in the chronic cases with out-growth of bone and lipping.

Schüller considers that the change in the contour of the joint in the group he calls polyarthritis chronica villosa is due entirely to the chronic inflammatory thickening and extensions of the synovial membrane caused by the invasion of a peculiar bacillus, and not at all to bony changes. The chief changes in the joint in the group he calls arthritis deformans are, on the other hand, in the articular cartilage, and depend on prolific over-growth of cartilage cells, swelling of cell spaces, and splitting up and wearing away of the cartilage at friction and pressure points, with out-growth of cartilaginous or bony tissue at the periphery. In this variety the synovial membrane forms villi, but these are less massive and much less vascular. They show the character of chronically inflamed connective tissue, and the bacilli present in the other form are absent.

Schüller confirms the observation that examination by Röntgen rays shows the absence of bony changes in arthritis chronica villosa and their presence in arthritis deformans. He regards the two affections as different in pathology and in clinical characters.

Examination with X-rays has shown that the bones in rheumatoid arthritis may look fragile, as if atrophied, and are more penetratable by the rays than is normal. Similar changes were noted by Adams from observations of bones after death.

Still describes the post-mortem appearances of joints in three children, the subjects of rheumatoid arthritis, at the Hospital for Sick Children. In two there was marked thickening and vascularity of the synovial membrane, capsule, and ligaments. Fibrillation of cartilage, eburnation of bone, and osteophytic out-growths were quite absent. The changes resemble those in the arthritis chronica villosa of Schüller.

Beyond the joint changes morbid anatomy shows little that is distinctive or constant.

The *muscles* show well-marked but not uniform atrophy, some fibres being more affected than others (Debove).

Peripheral neuritis has been present in some cases, and Pitres and Vailliard consider it is constant when there are trophic changes in the skin and muscles.

In certain forms of the disease in children adherent *pericardium* is found with striking frequency (Still), and its presence in adults also has been noted (Bannatyne, A. E. Garrod). Valvular disease is present in some cases, but hitherto its occurrence has been considered accidental, and as the result of previous attacks of true rheumatism.

Pathology.—The pathology of rheumatoid arthritis is a subject about which the most diverse views have been from time to time held by observers of repute.

Charcot and many French writers have believed that the affection has close affinities with true rheumatism, of which it is a chronic variety.

Jonathan Hutchinson regards the disease as the result of a combination of the rheumatic and gouty processes, in some cases one preponderating, and in some the other.

Arbuthnot Lane's views have already been referred to. He believes that the joint changes are due to merely mechanical causes—that they are essentially local and traumatic in origin. The theory that impaired nutrition of joints is the underlying factor in the production of the lesions characteristic of the disease has had wide acceptance.

Many writers have accepted the theory of an arthritic diathesis, by which is meant, as defined by Hutchinson, a bodily condition which renders a person liable, at some time and under suitable provocation, to become the subject of active arthritic manifestations.

Pye Smith believes the change to be of a senile nature.

Ord has referred the joint affection to peripheral irritation from such various sources as acute rheumatism, gout, traumatism, gonorrhœa, and utero-ovarian disorders, the cord being in a condition of excessive irritability due to anæmia, general exhaustion, or the climacteric. This theory regards the joint affection as the result of reflex irritation of the cord rather than of definite structural change.

That joint affections do arise in connection with gross cord lesions is shown by the occurrence of Charcot's disease in tabes, and by the trophic joint-changes in syringomyelia.

The presence of atrophy of muscles, often extreme, and other associated symptoms, such as abnormal sweating, pigmentation of skin, numbness and tingling, glossy skin, are important evidences of nervous influence.

Folli in 2 cases found some wasting of the cells of the anterior cornua of the cord, and in a few cases changes in the peripheral nerves have been seen. It is, however, a fact of overwhelming weight that no lesion of the cord or nerves has been found constantly which is capable of explaining the condition of the joint or muscle. Atrophy of muscles occurs also in other joint affections (tubercular and gonorrhœal), in which no question of a central origin is raised.

It has been suggested that the nervous phenomena are due to the effect on nerve tissue of toxins circulating in the blood produced by micro-organisms in the joints. The evidence on which this theory is based is briefly as follows:—

(*a*) That acute cases have points of resemblance to microbic diseases.

(*b*) That acute cases have followed closely some infective illness, such as scarlatina, influenza, acute tonsillitis, otitis media, suppuration behind a dental plate, bone disease, gonorrhœa.

(*c*) That micro-organisms have been found in the joints by Schüller, Bannatyne and Wohlmann, Chauffard and Ramond, von Dungern and Schneider, and others, which are considered by them to be the cause of the disease.

It is probable that previous infective diseases, such as those enumerated, "play some important part in predisposing the tissues to afford an entrance, as well as a suitable nidus for the development of the bacteria" (Bannatyne). If acute rheumatism comes to be included among the infective illnesses, as is in the highest degree probable, the significance of its occurrence in relation to rheumatoid arthritis would be rendered clearer.

If the origin of rheumatoid arthritis be bacterial it may be due—

(*a*) To a specific microbe in the joint, the secondary symptoms (atrophy, etc.) being due to the action of its poisons circulating in the blood.

(*b*) To an intoxication due possibly to a variety of infections, *i.e.* that of gonorrhœa, rheumatism, pyæmia, etc.

Bannatyne, working with Wohlmann and Bloxall, found a very minute organism in the synovial fluid, membranes, cartilage, and bony debris, and rarely in the blood. It is a bacillus measuring $2\,\mu$ to $6\,\mu$. It stains deeply at the two ends, the intervening portion remaining unstained. It may be stained by gentian violet, methyl violet, or carbolic fuchsin, but staining requires care. It grows best in peptone meat broth, and in culture has the appearance of gold dust. It grows also in milk, on agar-agar, and Loeffler's serum. Inoculation experiments have not been satisfactory. This organism has been found in no other joint affection, but has been found by Bannatyne in practically every case examined of rheumatoid arthritis of the acute polyarticular variety. Bannatyne did not find it in the chronic variety with lipping and bony outgrowth.

Chauffard and Ramond found a micro-organism short and thin, a diplo-bacillus, having much the appearance of a diplo-coccus. It stains with difficulty, and grows only in synovial fluid. Inoculation produces no results.

Von Dungern and Schneider obtained eighteen hours after death an organism from the fluid of the knee, wrist, and other joints, and also from the mucus of the gall-bladder. The organism was a small micrococcus closely related to B. lanceolatus. Injected into the knee joint of rabbits it produced lesions similar to those in the joints from which it was taken. The authors regard this organism as the chief cause of chronic arthritis in the case described, and believe that the infection was from a chronically inflamed gall-bladder. It took Gram's stain, and grew best on glucose bouillon.

Schüller finds in the acute form only—the form which he calls arthritis chronica villosa—a short bacillus with polar enlargement $2{\cdot}6\,\mu$ by $\cdot75\,\mu$ broad. It stains readily by ordinary methods, especially carbolic fuchsin, but decolorises easily. It grows on gelatine at 25° C., and on agar as a greyish film. Schüller finds this bacillus in the synovial tissue and fluid of the joint, and his results are chiefly obtained from living tissues removed by operation. It is present sometimes in company with micrococci, or with the gonococcus. He has not found the organism in the chronic form with bony outgrowth and lipping, for which he reserves the name arthritis deformans. Cultures of this bacillus have been injected by Schüller into the joints of rabbits, and he succeeded in producing the same villous formations in the synovial membrane which he describes in the human subject.

Schüller states that for the last fifteen years

he has examined the urine of patients with arthritis deformans, in the belief that the bone changes are connected with disturbances in the secretion of bone salts. He finds the calcium salts and earthy phosphates of the urine diminished. Further, he finds an increased amount of calcium salts in the cartilage of the affected joint.

Another view as to the source of toxins is that they are produced in the intestinal canal, especially the duodenum, as the result of fermentative changes, and exert a poisonous effect on the spinal cord, and through it on the joints and other tissues. This theory is well worth further investigation.

The rôle of auto-infection in disease is probably considerable, and in the intestine the formation of noxious substances becomes more abundant under a variety of morbid conditions. At present the larger number of the substances formed are unknown to chemistry, and the subject is one of great complexity.

The influence of septic conditions of the mouth deserves more investigation than it has hitherto received, and unhealthy states of the oral mucous membrane and gums are undoubtedly present in some cases.

This imperfect sketch of the present position of the pathology of the disease is a mere note to a mass of available material. It seems clear that further classification is necessary before the subject can advance towards solution. There can be no doubt of the importance in the production of the discase of such conditions of mind and body as tend to reduce vitality, and especially such as depress the nervous health. Depressing influences are probably always present in addition to others of a more direct kind.

If it be granted that organisms are a causative agent in the arthritis of certain types of the disease, the evidence of bacteriology up to the present time seems rather to favour the idea that the organism is not specific, but that the characteristic inflammatory changes may be brought about by more than one.

The valuable research of Poynton and Paine has gone far to establish the infective nature of rheumatic fever, and has identified a micro-organism which is capable of producing in animals the phenomena of the disease with greater exactness than in earlier experiments.

The subject of chronic arthritis has recently been under discussion by the Chelsea Clinical Society (*Lancet*, i. 1901), and also by the British Medical Association at the Annual Meeting at Cheltenham (*Brit. Med. Jour.* ii. 1901). Dissatisfaction with current theories as to the origin and classification of the various forms of chronic arthritis is apparent in the remarks of speakers in both discussions.

At the Pathological Society of London (*Brit. Med. Jour.* i. 1902), Poynton and Paine reported a successful intravenous inoculation made by them with a diplococcus isolated and cultivated from fluid in the knee joint of a man of sixty-seven affected with chronic arthritis, and who had died of carbolic poisoning. Arthritis without cardiac lesions was produced in two rabbits. This experiment takes one step further, the probability both that micro-organisms may be important factors in the production of rheumatoid arthritis, and also that more than one organism may be capable of contributing to the production of the chronic inflammatory changes in the joint with which we are familiar.

The *diagnosis* of rheumatoid arthritis does not in a well-marked case present great difficulty.

1. Chronic gout, *with crippling of joints as the result of repeated attacks of gouty arthritis*, may present some difficulty which inquiry into the history should dispel. The deformities of a chronic gouty joint may bear some resemblance to those of advanced rheumatoid arthritis, but in gout chalk-stones will usually be present either near the articulation or elsewhere in the body, and a definite history of acute attacks will be obtained. Gout is much more frequent in males, and is *at first* a paroxysmal disease, one or more joints being attacked acutely from time to time, with intervals of immunity, the great toe having a remarkable liability to early attack. Rheumatoid arthritis is progressive from the first, and even if its onset be acute, the joint affection does not clear up, but steadily increases from the time of attack.

Acute *articular* gout is a decidedly rare affection in women of the present day, but the immunity of even young women cannot be wholly relied on. The writer is acquainted with an acute case in a girl of nineteen.

It is of the highest importance for the patient's welfare that so serious a mistake should be avoided, for the principles of treatment in the two affections are wholly different, and nothing is more calculated to aggravate and confirm rheumatoid arthritis than to subject the sufferer from it to a régime suitable for the treatment of gout.

2. *Acute rheumatism* may be closely simulated by acute rheumatoid arthritis, and for the first few days the distinction may be impossible. The points which are most helpful are :—

(*a*) The joints attacked in acute rheumatoid arthritis are usually the small peripheral joints, and there is no tendency to rapid migration from one to another. Those first attacked remain affected, and soon show characteristic changes.

(*b*) Cardiac complications are less likely to occur, but are known. In adults this distinction is less valuable, as cardiac complications in true rheumatism are markedly less common in them than in children.

3. *Chronic rheumatism* is a condition ill-defined and variously understood. That most likely to be confused with rheumatoid arthritis

is the condition called by Jaccoud "rheumatism fibreux." His cases occurred after repeated attacks of acute rheumatism. Patients complaining of so-called rheumatic pains have usually a history of true rheumatism in themselves or their families, or are the subjects of rheumatoid arthritis.

4. *Gonorrhœal arthritis* presents some points of decided resemblance to rheumatoid arthritis. It does not migrate, but persists in the joint or joints first attacked, and the temporo-maxillary joint not rarely suffers. It is more common in men, and the larger joints are attacked rather than the smaller. Suppuration is rare, but not unknown, and there is a marked tendency to the formation of fibrous adhesions, but none to bony out-growth.

Acute myositis has been recorded in this affection, and iritis and conjunctivitis occur. In women a history of gonorrhœa is elicited with difficulty, and diagnosis has to be made by exclusion, if at all.

5. The extreme atrophy of muscles, in some cases out of all proportion to the joint affection, may lead to a diagnosis of progressive muscular atrophy. Ord mentions this possible resemblance. Payne thinks there is comparatively slight atrophy of the thenar and hypothenar eminences, and hollowing of the palm in rheumatoid arthritis.

6. Bouchard notes the occurrence of enlargement of the base of the second phalanx of the fingers in association with dilatation of the stomach, and considers this affection should be separated from rheumatoid arthritis.

7. Charcot's joint disease, the arthropathy of tabes dorsalis, is thought by some writers to have the same pathology as rheumatoid arthritis, the suggestion being that the disease is rheumatoid arthritis occurring in a patient profoundly debilitated by his cord disease. Clinically striking features are the complete absence of pain in the joint in association with degenerative changes so pronounced that its functions are in abeyance. Both knees may be full of fluid, and so disorganised as to form complete flails, yet no pain is felt, and the patient may even get about if the joint is fixed by an immovable splint.

8. The pain and rigidity due to involvement of the vertebral joints have been mistaken for spinal caries, and some care is needed in distinguishing between these affections, especially in cases where caries occurs late in life.

Prognosis.—The joint affection, once started, has no spontaneous tendency to recover. It is essentially chronic, and to a greater or less extent progressive in its course, the joint attacked being permanently injured or completely disorganised.

Treatment can do something to modify the unfavourable course of the illness, but to be effective it must be begun early and continued with untiring patience. When a joint is crippled and distorted by gross structural changes treatment can do but little to restore it to the normal; but if the disease be recognised early, before such changes have been produced, much can be done in many cases to prevent their appearance, and to ameliorate the condition of the patient. In some early cases the disease may be completely arrested.

Treatment.—Garrod laid down the principle that this disease must be treated by means (*a*) calculated to improve the general health, by a highly nutritious diet, and by tonics, especially such tonics as have a direct effect on the anæmia, and (*b*) by such local means as are calculated to improve the nutrition of the joints, and to prevent their becoming fixed and useless. Of a tonic treatment the importance cannot be over-estimated, and possibly a more hopeful view of the disease might be taken if it came under observation earlier, and if the treatment were always such as is suited for the arrest of the disease.

The *diet* should be as nutritious as the patient can assimilate, and should contain a sufficient quantity of red meat, in any form in which it can be taken, and of fats. A diet consisting wholly or chiefly of meat has been used extensively in this disease, and is considered by some to be of service. This diet is based on a work published in New York by J. H. Salisbury. Where the digestion of farinaceous food is difficult, and apt to lead to excessive fermentation, a diet consisting chiefly of meat may be useful, but it is not suitable to all patients, and some are quite unable to adopt it, and lose weight rapidly. Probably most patients will do best on a mixed diet.

Alcohol is useful in most cases in moderate daily amount, and with food. Sound red wines are the most suitable, but malt liquors are not contra-indicated as in gout, and stout is sometimes useful if it can be digested.

A low diet, and especially the limitation of red meat, is decidedly contra-indicated, and has an unfavourable effect on the course of the disease. The use of cream and butter should be encouraged.

Digestion and appetite are often impaired in the more debilitated patients, and feeding them suitably becomes very difficult.

Clothing should be light and warm—preferably of wool. It is important that the extremities, and especially the affected joints, should be carefully protected from cold.

Exercise should be persevered in so far as possible, though not to the extent of greatly increasing pain and fatigue. When exercise becomes difficult, the nutrition of the muscles of the limb should be kept up by rubbing or careful passive movements, and the patient should still be encouraged to use it as much as possible.

Fresh air is of benefit to these patients. They should live in a climate where outdoor life, without exposure to cold and damp, is possible. A dry soil, a high average of sunshine, slight daily variation of temperature, and a moderate elevation, are the most suitable climatic conditions. Winter in a warmer and more equable climate than England is desirable, but in the south of England localities can be found which are fairly well adapted to the needs of these patients. Sea-board and the beds of rivers should probably be avoided. Egypt suits well where the patient can travel without great difficulty.

As regards *drugs*, much benefit is derived from the use of tonics, iodine, and cod-liver oil, given over long periods. The most useful tonics are arsenic, iron, and quinine. Garrod laid stress on the great value of the iodide of iron in combination with arsenic. Arsenic should be persevered with steadily, with occasional intervals, and is perhaps more usefully employed in small doses for a lengthened period than when given in larger doses, which cannot be continued so long. The dried sulphate of iron in pill with arsenic is a useful combination. The iodides are of great value, but should be combined with tonics. Iodide of potassium alone has an unfavourable effect on the general health, which is not compensated by the relief it may afford to the joint pain. Cod-liver oil is highly beneficial, and patients should be encouraged to continue its use.

For the relief of pain hyoscyamus has been recommended.

Bannatyne and others, who believe that the acute forms of the disease have a bacterial origin, have employed with benefit drugs which are supposed to be antidotal to micro-organisms, or their products, when given internally. Bannatyne has employed the creasotes and guaiacols, the phenols (salol and salophen), and the naphthols, and believes that the creasotes have a more or less specific action on the disease, that the phenols are useless, and that the naphthols are of much use by reason of their effect on the contents of the intestine. He recommends the employment of carbonate of guaiacol, gr. v.-x. t. d. s. in cachet.

The salicylates reduce pain and fever in the acute stages, but should not be persevered with. Colchicum, invaluable in gout, is contra-indicated here.

Aspirin (salicylo-acetic acid) in doses of gr. x.-xv. relieves pain markedly, and can be taken over considerable periods without producing the depression which limits the usefulness of the salicylates in these cases. It is conveniently given in cachets, and is of decided value.

Ichthyol internally has been recommended, but reports are conflicting as to its value.

Baths have long been held in repute as a treatment for rheumatoid arthritis, and are of great value in properly selected cases. Baths are not indicated for the elderly and debilitated, who may be decidedly the worse for them, but benefit chiefly early cases, and those in whom the general health is fair, and where the joint affection has not advanced far towards hopeless deformity.

Baths necessitating long immersions are not advisable, and probably douche-massage, first practised at Aix-les-Bains, and admirably carried out there, is the most useful form. The exact chemical properties of the water employed is of less importance than skill and care in manipulation, but there is value in thermal water.

Bracing air is a decidedly valuable adjunct to treatment by bathing.

In this country Harrogate, Woodhall Spa, Buxton, and Bath have a reputation for the treatment of rheumatoid arthritis, and the three first named have the advantage of highly bracing air. At Bath the methods of Aix-les-Bains are carefully followed, and the water is thermal.

Llandrindod Wells and Droitwich are advised for these patients, and at Strathpeffer mud-baths are given as well as other treatment. The mud-baths of Ischl (Austria) are in high repute, and those of Acqui (N. Italy) have local renown. Acqui also possesses hot sulphur springs. Barèges (Hautes-Pyrénées) is a celebrated spa, but does not offer such luxurious accommodation to visitors as Aix. Wiesbaden is much frequented.

Where anæmia is marked a course of natural iron water at one of the ferruginous spas, such as Schwalbach, may be useful. The iron water of Flitwick is of decided value where digestion is weak and the ordinary preparations of iron disagree.

Electric baths have been much recommended. They should be employed only by skilled persons. The course needs to be long, two or three baths a week for two or three months. Such prolonged courses of baths would seem to be open to some objection. Turkish baths and vapour baths give temporary relief to pain and stiffness, but these baths repeated frequently are not beneficial and should be discouraged.

After a course of baths an after-cure spent in high and bracing air is of great benefit, and should, if possible, always be taken. In advising a course abroad it is well to warn patients to avoid the hottest months of the season. Baths during extreme heat may be followed by considerable exhaustion and decided increase of anæmia. One course of baths is rarely sufficient to effect the greatest possible amount of benefit. A second course should be taken after an interval varying according to the patient's condition, but never less than a year.

Heat locally to joints is a favourite treatment and has undoubted value. Probably dry heat is more efficacious than moist. A simple method is the hot sand bath; it can be employed wherever sand and an oven are available, and for the hand and wrist is specially useful. The limb is plunged into sand as hot as it can be borne, which is renewed as it cools.

Various methods of applying to affected joints dry air raised to high degrees of heat are in use. In the Tallerman method the heat is obtained from gas; the apparatus is of the nature of a gas stove, shaped so as to receive a limb. In the Dowsing and Greville methods the heat is generated by electricity, in the former by means of electric lamps, with reflectors of glass and metal, in the latter by means of small wires in a frame of aluminium. These methods need skilled application and special apparatus, and are necessarily expensive, but the relief obtained is in some cases very striking.

Massage is of value in keeping up the nutrition of muscles and preventing stiffness and fixation of joints. It relieves pain markedly in some cases. In association with the douche it is a most valuable treatment.

Cold has been recommended for the relief of pain, either locally in the form of ice-massage (Ewart) or by means of the spinal ice-bag. Ewart advises slowly rubbing the joint with a lump of ice once or twice a day, as a successful treatment when hot application fails.

Kinnear states that the application of the spinal ice-bag from half an hour to three hours a day relieved the worst cases when every known remedy had been tried and had failed. This author considers the ice-bag improves nutrition and circulation.

Electrical treatment apart from baths has been much recommended. Probably the constant current is useful in improving the nutrition of muscles. Static electricity is said to be of value as a general nerve tonic.

Surgical procedures have been employed in the following ways, though none but forced movements are at all generally used in this country:—

1. Operations on joints have been undertaken for the relief of ankylosis when the joint is fixed in a bad position, *i.e.* in the elbow or the temporo-maxillary joint. Collinson has had successful cases. He considers that operation is only applicable in young patients and where the muscles are not excessively atrophied.

Forced movements under an anæsthetic to break down adhesions are employed. They are not free from danger, and may set up acute inflammatory changes.

2. Schüller and others operate on joints for the purpose of removing diseased tissues, especially the synovial membrane.

3. Schüller has also treated cases by intra-articular injection of sterilised guaiacum-iodoform-glycerine. These procedures have for their object the removal or destruction of the organisms which the author considers are the cause of the arthritis.

External application of various medicinal substances is employed largely. Iodine painted on the joint or applied as an iodine plaster is useful. Tincture of capsicum applied on lint after washing the joint with hot soap and water may give decided relief. A liniment of chloroform, belladonna, and aconite in equal parts has been recommended. Bannatyne advises the use of equal parts of guaiacol and olive oil, or guaiacol with tincture of iodine (1-6), or guaiacol with methyl salicylate. The guaiacol is absorbed and can be detected in the urine. Its smell is a decided practical objection to its external employment, but it undoubtedly relieves pain in some cases.

Unhealthy conditions of the gums, especially such as are attended with purulent discharges, should be treated. Recent work makes it probable that chronic septic diseases of the mouth play an important part in auto-infection and the etiology of diseases which depend upon it. Affected parts should be cleansed thoroughly with 1-20 carbolic lotion, and an antiseptic mouth-wash used frequently; salicylic acid (1-600), or Condy's fluid well diluted, or Listerine, are satisfactory for this purpose.

Chronic Rheumatism

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Clinical Features.—The clinical features of chronic rheumatism are for the most part vague and ill-defined. The leading manifestations are seen in relation to joint structures, especially the larger joints of the limbs and spine, but many other tissues may be the chief sites of incidence of the rheumatic virus. Among these other manifestations rheumatic affections of muscles occupy a prominent place, and as these are very frequent and apt to be overlooked, special attention is devoted to them (p. 577). The skin, subcutaneous tissue, and the special senses of ear and eye frequently exhibit symptoms often regarded as of rheumatic origin. While there is a diversity of opinion on the relationship of these symptoms to rheumatism, the balance of evidence indicates an important

relationship. Among the diseases here referred to may be mentioned erythema, psoriasis, lichen, iritis, and chronic middle ear disease. In children many other subacute and chronic indications of disease are of rheumatic origin (*vide* "Rheumatism in Children," p. 550).

The clinical features of chronic rheumatism are perhaps best illustrated by short records of cases met with in practice. Those selected are characteristically common types, and it is needless to say that every grade of severity and various combinations of these types are met with in everyday practice. The probable relationship of these various types to each other will be referred to later under the etiology of the disease.

Mr. L., æt. 30.—Chronic myositis of the neck muscles; recurring arthritis in left hip and knee; deafness of middle ear origin (non-suppurative); acne and blind boils.

The hereditary history showed a distinct tendency to rheumatism. The joint trouble was confined to the left side, a feature which was probably correctly attributed by the patient to the results of a previous injury to the thigh, through being heavily thrown from a horse. The patient complained of a marked sense of discomfort in the head and neck, but had become accustomed to this feeling, and did not associate it with a local condition. Examination showed that the muscles at the back of the neck were unnaturally firm and contracted, the movements of extension and rotation of the head were restricted, the hip joint revealed creaking and limitation of movement; examination of the ear showed that the tympanic membrane itself was apparently normal, the structural changes in the middle ear were obviously of articular origin.

This case illustrates three interesting points—

(i.) A definite lesion in the muscles—chronic myositis (probably also cellulitis), a condition that is common and apt to be overlooked.

(ii.) The tendency to develop manifestations of rheumatism in a joint previously weakened, *e.g.* by a trauma, etc.

(iii.) The coexistence of the local disease in the ear with a general constitutional disturbance.

(iv.) A special liability to the pathogenic influence of bacteria on the skin surface.

Mr. M., æt. 46.—Chronic rheumatism in left shoulder joint; otherwise in apparently perfect health.

Mr. M. regards himself as in perfect general health. The only things that trouble him are his left shoulder and, occasionally, fleeting pains in other parts of the body. The joint condition has developed slowly, and has never been seriously treated. There is no distinct rheumatic history. The shoulder joint shows distinct muscular atrophy, limitation of movement, occasional creaking, and some irregularity at the head of the bone. Subjective symptoms are, as a rule, only complained of after a day's outdoor exercise, after which the left arm is for a time practically useless. There is no history of gonorrhœal infection in this case.

This case illustrates a very localised area of naturally weak power of resistance to the action of the rheumatic poison.

Mr. N., æt. 70.—Attack of subacute rheumatism when æt. 45; chronic arthritis in the knee joints since æt. 65; deafness of nerve origin.

Mr. N. leads a most active life physically and mentally; his general health is regarded by himself as excellent. He lives well, and has always done so. His joint disease makes him distinctly lame, but the pain complained of is inconsiderable, and always worst in the morning and on rising from a chair or couch. The right knee joint is most involved and is markedly enlarged, the swelling appearing to be largely confined to the soft tissues, especially in the anterior and superior aspects of the joint. There is well-marked creaking, but no new bony formations can be made out. The movement of flexion of the knee is restricted, and the hamstring muscles are strongly contracted.

This case is chiefly interesting as showing—

(*a*) An acute onset (when æt. 45) of a rheumatism which was *apparently* distinct from an ordinary attack of rheumatic fever.

(*b*) The association of a local disease in one of the special senses, along with the general manifestations.

(*c*) A fairly strong power of resistance to the virus of rheumatism, indicated by the limited nature and severity of the disease in a man of this age and history.

Mrs. O., æt. 60.—Rheumatoid arthritis, acute onset; chronic rheumatism.

At the age of 50 patient suffered from acute attacks of pain and swelling in the joints of the hands, ankles, and, to a less degree, of the wrist. The pain was evidently severe, as she was kept under morphia for some time. About the same time she began to complain of stiffness and weakness in the muscles in different parts of the body, a feature which persisted more or less for years, and is still present. Treatment by a change of climate, hydro-therapeutic measures, diet, and massage seemed to exert a favourable influence on the disease. Since the age of 55 the joint lesions have not progressed, but since that time she has had one severe attack of brachial neuritis and an acute attack of lumbago, and has been intermittently subject to marked pain and weakness in the limbs, and severe cramp in the muscles of the legs at night. At the present time there is considerable enlargement of all the joints of the hand, except the terminal joints, with distortion of the fingers, but no pain or limitation of movement. There is marked local muscular atrophy. One striking feature of this case which attracted the

patient's own attention was the state of the urine. It has persistently had a very strong odour, and was described by the patient as being frequently offensive. This patient is an interesting and by no means uncommon type of case, which illustrates the very close relationship that exists between rheumatoid arthritis and so-called chronic rheumatism.

Comparative Pathology.—Our knowledge of the clinical features, pathology, and treatment of rheumatism in the lower animals is in the same incomplete state as our knowledge of these affections in man. Horses, cattle, dogs, and pigs are known to be the subjects of disorders which in their clinical features present a close parallel to those affections in the human subject, and from a study of these diseases we may gain valuable information regarding the etiology of kindred affections in man. For our present purpose we may consider that the crucial question is, Do we find in the lower animals diseases which are clinically and pathologically identical with the following diseases as at present recognised in the human subject?—

(i.) Acute rheumatism.
(ii.) Chronic rheumatism.
(iii.) Rheumatoid arthritis or osteo-arthritis.

In answering these questions it will appear that veterinary surgeons have seen no reason to distinguish, or at least have not distinguished, chronic rheumatism and rheumatoid arthritis as two classes of disease. They simply differentiate them by the measure of severity in the involvement of the joints. The writer has devoted some attention to comparative pathology, and in addition to the knowledge derived from a direct clinical study of these conditions in the lower animals, he has obtained much information from available literature, as well as from friends in the veterinary profession.

(i.) *Acute Rheumatism.*—Animals are liable to an acute fever, characterised by a more or less sudden onset of high temperature, with painful enlargement of and effusion into the joints. The wrist and ankle are the joints most affected, and in them the pain is extreme. Metastasis is a prominent feature of this disease. Endocarditis and other cardiac conditions are frequent concomitants, and may lead to a fatal termination. The result may be (*a*) death from cardiac inflammation, (*b*) complete recovery, or (*c*) incomplete recovery, some local weakness being left in the affected joints. It is interesting to note that this disease is relatively more common in cattle than in horses. The analogy to acute rheumatism in the human subject is reasonably complete.

(ii. and iii.) *Chronic Rheumatism and Rheumatoid Arthritis.*—Symptoms attributed to chronic rheumatism and rheumatoid arthritis are extremely common in the lower animals, especially in horses, dogs, pigs, and cattle. We may say at the outset that, as less is known about the clinical features and morbid anatomy of these two conditions in animals than in man, it is necessary to discuss them together, merely submitting a brief outline of the clinical features and pathology on which their diagnoses are based. So far as I have been able to learn, the term rheumatoid arthritis, or more commonly osteo-arthritis, is applied to the cases which exhibit a more severe involvement of the joint, *e.g.* much irregular formation of new bone, muscular atrophy, with marked permanent disablement; while the term rheumatism is applied to a less severe articular lesion and to involvement of muscles, tendons, and tendon sheaths, bursæ, and nerves. The former class is naturally comparatively rare, as such animals are commercially useless, and are destroyed at a comparatively early stage of the disease, while in the less serious type of rheumatism, with less severe articular symptoms, the animal is able to do a fair amount of work. Thus we find that these latter cases form a not inconsiderable proportion of the subjects that come under the veterinary surgeon's care. It is convenient to consider this condition under the following headings:—

Articular Type of Rheumatism other than Acute.—(*a*) *Chronic.*—The disease is mainly, but by no means entirely, one of adult life and old age; cases in young animals, four and seven years, are by no means rare. The usual sites are the hip, spine, ankle, and wrist, but any joint may be involved. The clinical features are stiffness and pain in the limb, with swelling of the joint. The enlargement is due to an increase in the articular ends of the bones, or in the soft tissues entering into the formation of the joint. In some cases it is due to involvement of the bursæ or tendons in close proximity to the joint. Marked creaking may be elicited on movement of the limb. Investigation also may show that this animal never lies down at night, a symptom usually attributed to ankylosis of the lumbar spine. It is not an uncommon thing to find in a large stable of horses one or more animals in active work that are partially slung up at night so as to prevent them lying down. This precaution is advisable owing to their inability to raise themselves. Such an animal is very stiff in the morning, and may require a little exercise before being fit for even a light day's work. The creaking heard on movement may be due either to disease in the joint or in the muscles surrounding it. The muscles and tendons on the flexor aspect of the joint are usually unduly hard and contracted. In mild cases, when the animal gets warmed to his work, all stiffness and lameness may disappear, and nothing further may be determined about the case except that he is subject to constipation, and voids urine that is turbid and evidently unduly charged with salts of various kinds. A careful examination into the history will usually reveal the fact that the animal

drinks too little, or, to put it more accurately, he does not get the opportunity of drinking enough. Here, then, we have a clinical picture of disease, the prototype of which comes within daily observation of the medical practitioner.

(*b*) *Subacute.* — Subacute manifestations of rheumatism are extremely common, and in this group we find the most striking illustrations of metastasis. These metastatic manifestations are frequently of an acute nature, with pain, effusion, and elevation of temperature, but to a much less degree than is characteristic of the acute cases previously described.

(*c*) *Abarticular Type.*—This group includes acute and chronic cases. The former are characterised by acute pain and swelling of the tendons and other soft tissues in the neighbourhood of a joint. The symptoms may be so severe as to prevent the animal rising for several days. This type of disease is not, as a rule, accompanied by any febrile reaction. The chronic group includes many cases of "spavin," "ringbone," and other conditions characterised by local formation of new tissue of a bony or other nature in the neighbourhood of joints. Mention must also be made of cases of lameness that are supposed, by a process of exclusion, to be dependent on a neuritis of a "constitutional" or "rheumatic" nature.

(*d*) *Rheumatoid or Osteo-Arthritis.* —As already indicated, this term is applied to the more severe type of cases of chronic rheumatism, when the articular changes are more advanced and of a more markedly hypertrophic nature. The subjects in this group show greater muscular weakness, and usually advanced local muscular atrophy. It is important to note that this disease is sometimes well developed in quite young subjects; a few cases have been recorded from zoological collections.

It will be seen from the foregoing that, so far as clinical features are concerned, the diseases of a "rheumatic" origin in horses correspond very closely to the same diseases in the human subject. What differences exist are probably of degree rather than of kind, and are readily explained by the different life-histories of the two subjects. When we turn to the pathology we will find that the comparison holds good.

Pathology.—The writer has examined many of these cases (groups *a* and *d*), and has found that the joint changes are very similar to those encountered in cases of chronic rheumatism and rheumatoid arthritis in man. In many cases the description of joints in chronic rheumatism (vol. iv. p. 242) may be fitly applied. In others the appearances more closely resemble those recorded for osteo-arthritis in its earlier stages (vol. viii. p. 567), and also the lucid description of traumatic arthritis given by Arbuthnot Lane. So far as the writer's observations go, the morbid anatomy hardly warrants any distinct classification. The differences appear to be of degree rather than of kind. This subject is further referred to under etiology.

Treatment.—It is beyond the province of this article to refer to treatment except in one particular. We have already seen that clinical experience and pathological investigation show a close parallel between these affections in the lower animals and in man. Confirmatory evidence is obtained from the field of therapeutics. To speak generally, veterinary surgeons treat these affections in one of two ways: (*a*) by rest and the adoption of local measures, mainly of a counter-irritant nature, and (*b*) by a limited amount of exercise, a laxative diet, and plenty of water. The balance of opinion is in favour of the latter course—a fact not without its lessons to the medical practitioner. With regard to diet, if space had allowed, many interesting facts could be quoted regarding "chronic rheumatism" in other animals, *e.g.* pigs, and the relationship of diet to that condition.

Morbid Anatomy.—The naked eye features of a chronic rheumatic joint in the human subject are very similar to those met with in the lower animals. In both the joints are unnaturally dry; when fluid is present, it is altered in colour and consistence. There is the same tendency to irregular enlargement of the articular ends of the bones. The tendons and tendon sheaths in the neighbourhood of the joints show the same congestion of vessels and evidences of recent and old cell proliferation. The cartilages may be thickened or thinned, and may show vascularisation and ulceration. The synovial membranes show a varying degree of thickening and undue vascularity, and in some cases irregular villous projections. There is a further correspondence between the two classes of rheumatic subjects in that no two cases, and even no two joints in the same case, present exactly the same appearance. This close parallelism, however, is of little value for the etiology of the disease. The gross structural changes which take place in the affected joints must be set in relation to the other changes induced in the tissues, and it is probable that the facts of a more general morbid anatomy will prove of much greater importance for the discovery of the cause of chronic rheumatism than the facts of those articular changes to which attention has been too exclusively confined.

With regard to the remaining vital constituent of the morphological joint unit—the marrow—I know of no observations on the human subject as to the state of this very important tissue in this disease. In the course of an examination of many subjects in comparative pathology in which symptoms of rheumatism had been present, the writer has frequently seen definite gross changes in the bony system other than the articular ends. These changes were mainly of the nature of (*a*) altered colour and appear-

ance of the marrow, a varying degree of redness and gelatinous degeneration being present; (*b*) altered consistence of the marrow due to sclerosis and calcareous changes in that tissue. Owing to an insufficient acquaintance with the normal appearance in animals at different periods of life, it is not possible to speak with assurance as to the character of these pathological changes and their relation to the general rheumatic condition. But as to the existence of gross changes there is no room for doubt, and, to my mind, little reason for doubting that these have a close etiological relationship to the disease itself. This field of study is a wide and important one. Till now, the pathological histology, like the morbid anatomy, of these cases has taught us little about the cause of chronic rheumatism, and until we have given adequate study to the bone marrow and its function, we shall be unable to formulate with confidence a theory as to the etiology of the disease.

A further desideratum in human pathology is an exhaustive examination of the naked eye and microscopic appearances of all the viscera and tissues in cases either of rheumatism or so-called rheumatoid arthritis. In both classes of case we are too ready to conclude that visceral lesions, if present, are relatively unimportant or incidental complications. What is desirable to have even in one case of this kind is a complete histological examination of all the tissues of the body. Very special attention should be directed to the following:—

(*a*) *Very detailed examination of the vessels*—small and large—concerned in the nutrient supply of the joints, muscles, nerves, or other tissues that were the seat of the clinical manifestations. Attention has lately been drawn to the presence of disease in the arteries and capillaries in some cases of acute rheumatism (*see* p. 539); it is probable that similar local changes of a more chronic nature may help to explain some of the fleeting manifestations of chronic rheumatism.

(*b*) Similar *examination of the various mucous surfaces* that are the common sites of infection for bacterial invasion—more especially the gastro-intestinal canal and the utero-vaginal membranes. The facts learned from this examination would be many, and could not fail to be very instructive.

(*c*) The naked eye and microscopic *appearance of the marrow.*

With these facts and the others obtainable from present records, we would be in a better position to review the whole picture of rheumatism, and would also be in a stronger position for formulating a theory regarding its pathogenesis. But these facts are absolutely essential.

Rheumatism, Muscular.—In rheumatic subjects the muscles are affected in two ways—as an acute or a chronic myositis. Both conditions are apt to escape notice on account of the small amount of attention that is paid to the systematic clinical examination of the muscular system. It is sometimes stated that where there is evidence of active inflammatory change in the muscles the condition is to be regarded as myositis, and no longer as muscular rheumatism—the latter name being reserved for those cases in which no objective signs are present. This is surely an erroneous view of the subject; there is much evidence in favour of the view that so-called muscular rheumatism stands in a very direct relation to other manifestations of rheumatism, and a study of the manner in which the muscles are affected is essential to a complete survey of rheumatism.

Acute Myositis.—Acute myositis is met with at any period of life, but is more common in the later half. The female sex is more liable, and stout subjects especially so. The clinical features vary according to the extent and severity of the disease. The patient may complain of "neuralgia," or of "rheumatic pains" in a joint, or of weakness in a limb. Little pain may be complained of when at rest, but on exertion, *e.g.* rising from a chair, an acute twinge of pain is experienced. In cases of some duration and severity the amount of discomfort or pain is considerable, as are also the pains of the limbs as a whole, and especially the weakness in the joint nearest the muscles involved. On examination of the affected muscle nothing will be found unless a most careful and detailed examination, especially adapted to determine the presence of inflammatory thickening in the muscles, is carried out. The examination is made as follows:—The patient must be directed to put the muscles into a passive state, as a contracted state of the fibres will interfere with the examination. The skin is smeared with vaseline. The muscle is then gently palpated with the finger-tips, the examination being conducted along as well as across the line of the fibres. The movement of gentle petrissage is well calculated to reveal the presence of foci of rheumatic inflammation. A patch of inflammation reveals itself by marked localised tenderness, which makes the patient wince keenly. The painful spot is usually the size of a bean, but may be smaller or considerably larger. To the finger the spot feels hard in the centre and surrounded by an area of unusual softness, presumably an area of serous exudation. These spots are usually multiple, and a careful examination may reveal their presence in regions unknown to the patient. The muscles most commonly affected are those on the front and sides of the chest (pleurodynia), thighs, neck, legs, and abdomen, but practically all the muscles of the body may be involved.

Chronic Myositis.—The typical picture of a case of chronic myositis is quite distinct from the foregoing, but here also the clinical features

are not such as direct attention specially to the muscular system. The patient may complain of—

(i.) Rheumatism, involving mainly one or more of the larger joints. It is advisable to note that an objective examination may reveal definite articular changes which distract attention from the more important lesion in the muscle.

(ii.) Neuralgia.—Especially of neuralgic pains in the occipital region or back of neck, thighs (sciatica), or lumbar region (lumbago).

(iii.) Headache and general discomfort in the head and neck are sometimes the leading symptoms complained of.

(iv.) Precordial pain, or pain in the mammary region.

On objective examination the affected muscles are found to be in a state of contraction. They are unduly hard throughout their whole extent, and when their action is tested, their movement is found to be unduly limited and markedly painful. In the affected part there may be none of the acutely painful areas characteristic of the acute condition, but careful examination may reveal their presence in another region of the body. The muscles most involved are the same as those already mentioned. Special mention should be made of the muscles attached to the occiput and neck, to the intercostal and serrati and gluteal muscles.

Diagnosis.—The differential diagnosis is, as a rule, easy, provided a careful examination of the muscular system be carried out. The following diseases are simulated, and muscular rheumatism may be mistaken for—

(*a*) Joint disease.—Pain and stiffness is often referred to the joint, which may be in a state of flexion. This is especially seen in the knee, hip, and shoulder.

(*b*) Visceral disease, especially pleurisy, or some disturbance in the gastro-intestinal tract, *e.g.* gastralgia, appendicitis.

(*c*) Cardiac disorder or disease of the mammary gland in women. Symptoms attributed by the patient to these conditions are not infrequently due to rheumatic inflammation in the muscles of the chest wall.

(*d*) It may not be possible to differentiate myositis from similar small painful nodules met with in the subcutaneous tissues due to a *local cellulitis*, which is practically identical in its origin and nature with the areas of inflammation in the muscular tissue. From a practical point of view, an accurate diagnosis is immaterial.

It is only necessary to mention the presence of small subcutaneous fibrous nodules sometimes met with in the subcutaneous tissue or attached to tendons, seen especially in the forearm. These are apparently small fibrous nodules of local origin, and do not call for treatment.

(*e*) *Neuralgia.*—The frequency of these distinct inflammations in muscles is probably not sufficiently realised, and not a few cases are diagnosed as "neuralgia." The writer believes that some cases diagnosed as brachial neuralgia are really cases of myositis involving the muscles of the neck; occipital neuralgia, due to inflammation of the deep muscles, especially the complexus; sciatica, due to local changes in the glutei muscles, and some cases of lumbago due to a similar state in the lower fibres of the trapezius or deep muscles of the trunk.

In the more chronic cases, *e.g.* in the neck, it may be quite impossible to say whether the structural change is in the muscles of the part, in the subcutaneous tissue, in the nerves, or in the joints of the spine. Some assistance in the diagnosis may be forthcoming from the result of treatment. After one day's treatment by gentle effleurage the skin over the inflamed portion becomes yellow, and later assumes a darker tint, the colour disappearing in three to four days, and by that time the affected limb, which previously felt heavy, dull, and paralysed, feels alive and in a much improved state.

General Diagnosis.—Some important points in the diagnosis of rheumatism have already been detailed in the section on "Muscular Rheumatism."

Firstly, It is advisable to determine as far as possible the exact site of the pathological process. Is it in the muscle, subcutaneous tissue, nerve, or in the neighbouring joint? The accurate determination of this point may materially modify the prognosis in a given case.

Secondly, The existence of any gross visceral disease ought next to be determined by an examination in turn of the gastro-intestinal, renal, cardio-vascular, and nervous systems. In women special attention should be directed to the menstrual function. When gross visceral disease is detected, we should not at once consider that such is a merely incidental complication of the rheumatism, as in most cases it is probably closely identified with it, etiologically, pathologically, and clinically.

Thirdly, In doubtful cases of articular rheumatism it is not necessary to differentiate precisely the various types of osteo-arthritis, as the relationship between that disease and so-called articular rheumatism is probably a close one, and the principles of treatment are essentially the same.

Fourthly, With regard to gout—what has been said of articular rheumatism holds good also with regard to gout. It is sometimes said that great care is necessary to differentiate cases of joint disease, the result of chronic gout, from cases to which, on clinical and pathological grounds, the term osteo-arthritis appears more applicable, as the dietetic and general treatment is so different in the two cases—the first calling

for depletory, the latter calling for more stimulating measures. But this difference in treatment is only sound in a restricted sense, *i.e.* with reference to the later stage of the disease, when we are treating the results of the disease. In the early stages of both diseases the principles of treatment (*vide* article "GOUT," vol. iii.) are the same.

ETIOLOGY.—The etiology of chronic rheumatism is quite unknown, and we do not possess evidence sufficient to warrant a definite theory being put forward for the causation of this disease. We have already pointed out the advisability of obtaining new facts which might materially advance our knowledge, viz. (*a*) the state of the vascular system—especially arteries and capillaries—in the affected tissues; (*b*) the histological appearances of the various free mucous surfaces in the body—especially the gastro-intestinal tract and utero-vaginal mucous membrane; and (*c*) fuller and more accurate information on the naked eye and microscopic appearance of the bone marrow. If to these we were to add the results of an investigation into the combination of various gross and minute changes in the renal, cardio-vascular, and nervous systems with chronic rheumatoid and rheumatic changes in the same subject, I believe a new chapter on the etiology of the disease could be written. And if we were to avail ourselves more of the wealth of clinical material obtainable from diseased subjects in the lower animal kingdom a great advance in our knowledge would be assured; for the study of these subjects can be conducted with greater ease, and is productive of results at once fuller and more prccise, than is possible in the case of the human subject. In the absence of this necessary information we shall only refer briefly to some of the views in vogue regarding the relationship of these diseases to each other, and thereafter merely mention a view which seems to present at any rate a *prima facie* case for a careful consideration. Three critical queries suggest themselves:—

Firstly, Is it wise to accept the general tendency at the present time to regard rheumatoid arthritis and true rheumatism as maladies essentially distinct and widely differing in their pathology?

Secondly, In our zeal for classification and recognition of types, may there not be a tendency to fail to recognise certain general pathological principles underlying all these diseased processes?

Thirdly, Should we regard "muscular rheumatism" as a disease *sui generis*, not closely connected etiologically with articular and other manifestations of rheumatism?

There would appear to be many facts which suggest that acute rheumatism, chronic rheumatism, and rheumatoid arthritis are in reality closely related to each other, the varied clinical and pathological pictures merely representing different individual reactions to the same cause or group of causes. The grounds for this belief can only be briefly stated—

(*a*) In practice there are frequently extreme difficulties in differentiating between acute rheumatism and acute polyarticular rheumatoid arthritis. In not a few cases the diagnosis is impossible.

(*b*) It is well known and generally recognised that a typical pathological picture of rheumatoid arthritis may develop as a sequel of infection of the system with the virus of gonorrhœa and, more rarely, of other pathogenic bacteria.

(*c*) A frequent investigation of all the cases of a rheumatic or rheumatoid nature in the hospital wards of a large workhouse has convinced me of what may be termed their hybrid nature in a very large proportion of the cases seen.

(*d*) We find a good deal of evidence in favour of the idea of some unity in the causation of these diseases in the writings of living authorities on these special departments. Thus it is admitted "that there are cases in which deformity results from acute rheumatism and remains permanent. Such cases, though they resemble rheumatic fever in their varied manifestations, are in the character of the changes in the joint more akin to rheumatoid arthritis" (Poynton). In his article on rheumatoid arthritis in children (*System of Medicine*, Allbutt, vol. iii. p. 106), Still writes as follows: "Chronic fibrous rheumatism seems to be the best name for a third disease which has probably been indiscriminately included under the term rheumatoid arthritis in children. Its etiology and pathology are probably identical with those of acute rheumatism"; and in the article on Chronic Rheumatism in the same volume, Garrod, after referring to the difficulties of nomenclature and classification, states—

"We may group the cases to which the name of chronic rheumatism is more or less correctly applied under three heads as follows:—

"(i.) Frequently recurring slight articular attacks, in rheumatic subjects.

"(ii.) Cases of chronic abarticular rheumatism, a condition best seen in children.

"(iii.) Joint lesions which persist after an attack or succession of attacks of acute or subacute rheumatism, and in some cases lead to permanent damage of the structures assailed."

These expressions of opinion taken collectively seem to harmonise with the teachings of clinical experience as to the etiological relationship between the different manifestations of rheumatic and rheumatoid disease in man, and they certainly lend colour to the suggestion that all these diseases may be due to the same cause or group of causes.

The known facts of bacteriology prove that

acute rheumatism is of bacterial origin, but do not admit a final statement being made as to the single nature of the micro-organism. A good deal can be said in favour of Singer's view (p. 539) as to rheumatic fever being an attenuated pyæmia, and it is possible that the various subacute and chronic manifestations are susceptible of a similar explanation. Certain known facts lend some support to this opinion. Thus we find that Alexis Thomson, in the article on Diseases of Bone in vol. i. p. 426, writes as follows:—

"The marrow, especially in the interior of long bones during the period of active growth, is known to be one of the most favourable of soils for the lodgment of bacteria. The latter may persist in a condition of potential vitality for indefinite periods long after they have disappeared from the circulatory blood from which they were derived. The determination of disease in the bone resulting from bacterial lodgment in the marrow will depend, both as to time of onset, severity, and extent, on the initial virulence and number of the organisms, and upon the resistance of the tissues of the individual concerned, and upon the assistance of certain external conditions, *e.g.* trauma, strain, exposure, and cold, which will act also as localising factors in determining the seat and gravity of the disease." The same authority also refers to an attenuated form of staphylococcus osteo-myelitis, or growth fever (vol. i. p. 437), regarded by some as the explanation of "growing pains" in children and adolescents, and points out that definite changes have been found in the marrow in this disease.

The varying sites of incidence in the muscles, joints, heart, etc.—the different degrees of severity of the local manifestations — the presence or absence of febrile, and other general constitutional phenomena,—are probably all susceptible of explanation if we keep clearly in view the fact that no two people react alike to the same toxic agent. A study of the individual factor in disease is of paramount importance. And we must further keep in mind that the age of the patient probably influences that reaction materially, the tissues in childhood and early life reacting more strongly than at a later period. There is one point in the bacteriological aspect of the question that calls for reference. Investigations on the bacteriology of chronic rheumatoid arthritis have given uniform results only, in so far that great difficulty has been experienced in reproducing the disease in lower animals.

In this connection we must bear in mind the chronicity of the disease, and the possibility of attenuation of the bacteria at the diseased foci. The writer has lately examined the tissues of a horse seventeen years old, which had led a fairly active life for twelve years, during which time there had existed a small sinus in the groin, the result of sepsis at castration. Post-mortem examination showed widespread gross and microscopic lesions—the lesions in the kidney being very characteristic of a chronic pyæmia; but inoculation experiments carried out by an expert have not been successful in inducing any pathological lesions in lower animals, a fact which may be due to attenuation of the bacteria. This point is mentioned as worthy of consideration in connection with experimental research on chronic diseases in man.

With regard to the general causes of chronic rheumatism we need only refer to such conditions as exposure and cold, damp, the influence of a soil with a low-level water, and exposure to other infections, *e.g.* influenza. All of these are of a nature favouring bacterial invasion.

Treatment.—*Prophylactic.*—As the poison of rheumatism in its various subacute and chronic forms is one which specially selects the muscles and the soft and bony tissues entering into joint formations, the leading indication is to keep the tone of these structures, and especially the muscles, in as good a condition as possible. This sounds a truism, but as such it merits more attention than it generally receives. More attention to systematic physical education of youth is the basis of a rational prophylactic treatment. The value of this line of treatment is one which is readily demonstrable. There are many people who consider themselves in good health although they suffer from rheumatic pains and aches to a considerable extent. If these persons take a systematic course of exercises adapted to accelerate the circulation, and otherwise improve the tone of the muscles and nerves involved, in a very short time they will experience very marked relief from these manipulations—the permanence of the benefit being directly proportionate to the duration of the treatment.

This principle of prophylaxis is well illustrated in many Continental health resorts where the English and other visitors take a course of treatment in one of the Zandar institutes. This excellent plan could sometimes be followed by better results if the course were taken in a less haphazard fashion than is often done.

Curative.—For the curative treatment it is more advisable to lay stress on the principles which must guide us rather than on the means by which they are to be given effect to. If there is any breach of surface, *e.g.* cystitis, endometritis, and the like, special attention must be directed to that, as also to the state of the gastro-intestinal tract, and we must bear in mind that a considerable amount of disease may be present in the mucous membranes without revealing itself by any definite clinical symptoms.

(*a*) *Attention to the Excretory Organs.*—The

intestinal function should be promoted by the administration of a laxative diet and judicious use of aperients. If the bowels move naturally every day all that is advisable is the administration of a small blue pill, followed by a morning saline once in ten days or so. If further treatment is indicated, as is usually the case, then the selection of a suitable aperient is the next point to be attended to. The best is perhaps the judicious combination of vegetable aperients, *e.g.* aloin, euonymin, with occasional saline. But in connection with these points we should remember that the constipation is more likely to be indirectly improved by the physical treatment later referred to. The aperients are merely adjuvants. In many cases there is marked fœtor of stools, a fact of considerable significance, calling for appropriate dietetic and medicinal treatment.

Renal Function.—The renal function is to be favoured by the patient drinking more fluid than is his wont. The best fluids are water and alkaline drinks. These are preferably taken on an empty stomach, the amount varying from about one pint more than his usual. We sometimes find that the patient never drinks anything except at meal hours, when the fluid taken is therapeutically not so beneficial as those just mentioned.

The skin function is best promoted by hydrotherapeutic measures, which must in every case be adapted to the strength of the patient. Above all, excess in this direction is to be avoided (*see* "Balneology," vol. i.). For convenient use at home, one of the numerous bath cabinets now in vogue can be recommended; these require judgment and care in their use.

By the foregoing we may hope gradually to eliminate some, at least, of the toxic ingredients in the system in these cases, and we now pass to consider the means to be adopted in order to prevent further ingestion of substances likely to be injurious to the organism.

Physical Therapeutics.—Having discovered so far as possible what joints, muscles, tendons, or nerves are specially implicated, due attention must be paid to the affected parts. One precaution is specially important. The practitioner must be fully satisfied that the assistant whom he employs for this purpose has had a really expert training in his profession, and not merely a few months' fragmentary education. Assistants of the latter type often do more harm than good, and their extensive employment is one of the reasons why treatment by manipulation, massage, etc., has not its due place in therapeutics.

Diet.—If the bowels are thoroughly attended to, many cases require no dietetic treatment, provided the diet taken is an ordinary one. In many cases the patient is aware that certain articles of diet affect the rheumatism unfavourably, and the use of such foods or drinks should be avoided or restricted. Nature is the best guide in these matters. Some rheumatic subjects keep themselves comparatively free by a vegetarian diet. Our present knowledge of the etiology of rheumatic affections does not allow of any hard and fast rules *re* diet. The following are suggested by clinical experience:—

(i.) The diet should be a simple one, taken at regular intervals and eaten slowly. When the digestion is good and the appetite strong, a large amount of food may be taken, provided it be of a simple nature.

(ii.) The articles of food and drink which patients consider detrimental to themselves should be withheld or given in sparing amount. Their nature may give a clue to the direction of faulty metabolism and assimilation. In this connection it is well to note the periodical nature of the symptoms, *e.g.* the recurrence of rheumatic symptoms at the fruit season.

(iii.) More fluid should be drunk. Most rheumatic subjects drink too little water. The writer lately saw a stout lady much affected with chronic rheumatism. Investigation elicited the fact that she never drank anything except a little tea twice or thrice daily, and a little whisky and water at night. The total urine was collected for three successive days, and was as follows:—8, 14, and 10 oz., of a non-albuminous urine of average sp. gr. 1015. The patient avowed that this was her usual habit, and it is probable that many rheumatic subjects suffer from this anuria in varying degrees.

(iv.) If the patient can furnish no indications as to diet, the best plan will be—(*a*) simplify the food by cutting off some of the unnecessary ingredients in the daily diet; and if that is insufficient, (*b*) restrict the carbohydrates, *e.g.* sweets of all kinds, potatoes, and other underground roots, and all puddings of a "stodgy" nature. A few cases may do better on a more strictly nitrogenous diet.

Medicinal Treatment.—Medicines are useful, but should occupy a subordinate place. A blue pill occasionally, with free use of alkaline and mineral waters, is a valuable line of treatment, which probably acts mainly by improving the conditions of the gastro-intestinal, hepatic, and renal functions. The old-time remedies of sulphur—internally and externally—and guaiacum are also very serviceable. Salol, salicylate of soda, and the like remedies are of service in some cases, and a judicious combination of these with other remedies possessing some intestinal disinfectant action are materially useful in some cases. The same can be said of the internal administration of iodide of potassium, and the internal and external use of various fir oils (*see* also "Balneology," vol. i., and "Gout," vol. iii.).

Rheumato-.—In compound words *rheumato-* means rheumatic or relating to rheuma-

tism; *e.g. rheumatodynia* (rheumatic pain), *rheumatospasmus* (rheumatic cramp), *rheumatorchitis*, etc.

Rhinencephalon.—The olfactory bulb, "the most archaic part of the hemisphere" of the brain; in the human subject it becomes solid after the first month of embryonic life, and is represented in the adult by the olfactory bulb, the olfactory tract, the uncus, the septum lucidum, the fornix and hippocampus, the gyrus dentatus, etc.

Rhineurynter.—An apparatus (bag capable of being inflated) with which the anterior nares may be plugged in cases of epistaxis.

Rhinitis. *See* Nose, Acute Inflammation (*Rhinitis*); Diphtheria (*Clinical History, Nasal Diphtheria*); Syphilis (*Tertiary, Nose*).

Rhino-.—In compound words *rhino-* (Gr. ῥίς, ῥινός, the nose) means relating to the nose, nasal; *e.g. rhinocleisis* (nasal obstruction), *rhinocnesmus* (nasal itching), *rhinodynia* (nasal pain), *rhinolaryngitis* (inflammation of the nasal and laryngeal mucous membranes), and *rhinorrhagia* (nasal hæmorrhage).

Rhinocephalus.—A teratological type in which cyclopia (fusion of the eyes) is associated with the presence of a nasal proboscis situated above the single orbital cavity. *See* Teratology.

Rhinodyme.—A teratological type in which there are two noses, more or less fused together, along with duplicity of the superior maxillary bones. (*See* Ballantyne's *Antenatal Pathology*, vol. ii. pp. 233, 422, 1904.)

Rhinolalia.—A defect of articulation due either to incomplete closure or incomplete opening of the posterior nares. *See* Pharynx, Examination of the (*Neuroses, Paralysis of the Soft Palate*).

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